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VOL. 46 MAYO. 1954 NO. 5

INDICE DE MATERIAS

Página

Entered as second class matter, January 21, 1931 at the Post Office at San Juan, Puerto Rico, under the act of August 24, 1912.

Hematímetro Bright-Line Spencer

El Hematímetro Bright-Line Spencer es un instrumento de muchos usos: para las cuentas de levadura; para las cuentas de polvo; para las cuentas en los fluídos espinales, salivales u otros fluídos del cuerpo. Sin embargo, su uso principal es para realizar cuentas de células rojas y blancas de la sangre. Para esa tarea se ha hecho la norma en muchos hospitales y laboratorios clínicos, resultando una ayuda imprescindible para muchos miles de médicos y estudiantes.

La característica singular y más notable de la Cámara Contadora Bright-Line es el contraste muy distinto de las líneas brillantes contra el fondo metalizado más oscuro.

Visibilidad Superior por Contraste

Una eficiencia mayor y más comodidad resultan por el uso de este Hematímetro, con menos fatiga y tensión ocular, porque la visibilidad resulta superior a la de otras cámaras. Bajo el microscopio, las líneas parecen blancas, contra el fondo semi-transparente más oscuro, con los corpúsculos echados en relieve muy distintivo. La visibilidad de las líneas depende mayormente de ese contraste. Las líneas triples de división muestran claramente cuáles células descansan dentro de las áreas para cuenta.

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PUERTO RICO OPTICAL COMPANY

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For soundness in the infant's formula . . . check all

Sound example of three-dimensional structure is LACTUM, Mead's evaporated whole milk–Dextri-Maltose® formula.

1. Sixteen per cent of Lactum's calories are supplied by *milk protein*—providing a generous amount for growth and development.
2. *Milk fat* contributes 34% of the calories.
3. *Carbohydrates* supply 50% of the calories—to provide generously for energy, permit proper metabolism of fat, and spare protein for tissue-building functions.

Authoritative pediatric recommendations support this caloric distribution. And cow's milk and Dextri-Maltose formulas with these approximate proportions have been successfully used in infant feeding for forty years.

Lactum's 4th dimension . . .
Time-saving convenience

Simply add water. A 1:1 dilution of Lactum provides 20 calories per fluid ounce.

FOR INFANTS WITH LOW FAT TOLERANCE

DALACTUM, an evaporated low fat milk and Dextri-Maltose formula offering the same convenience as Lactum.

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MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U.S.A.

P. O. Box 3081 — San Juan, P. R.

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. . . En el Polo Norte o en la Selva Tropical

En cualquier clima, bajo las condiciones más variadas, la Leche KLIM es siempre segura, pura y uniformemente nutritiva. En efecto, KLIM ha sido usada en muchas expediciones al Polo Norte y al Polo Sur y bajo el sofocante calor de las selvas ecuatoriales. Y siempre la frescura uniforme, sabor y calidad inmejorables de esta cremosa leche en polvo permanecen intactos en su lata cerrada al vacío, envasada por medio de un procedimiento especial.

Considere también, Doctor, otras grandes ventajas de la Leche KLIM, tal como su fácil digestibilidad, una de las razones por la cual muchos pediátras recomiendan esta leche superior en la alimentación de los niños.

Además de que es una leche ideal para la nutrición de los niños y para la familia en general, KLIM es un alimento suave pero muy vigorizante para los ancianos de estómago delicado. Esta leche tan saludable también se recomienda en las dietas de convalecientes y frecuentemente en las de aquellos que padecen de úlceras pépticas.

No hay mejor leche que KLIM.

KLIM

LA PREFERIDA EN TODO EL MUNDO

Nos complaceremos en suministrarle informes profesionales completos sobre la Leche KLIM. Sólo tiene que escribir a:

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For adequate nutritional supplement, B-NUTRON supplies B-*Complex* and *Iron* in a palatable, non-alcoholic syrup, easy to take by patients of all age groups.

B-NUTRON TABLETS
supply essentially the same formula – for adults who may prefer tablets.

℞ 4, 8 or 16 oz. – Syrup
40's and 100's – Tablets

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Un **nuevo** *y superior*

Antibiótico

de amplio espectro

Acromicina*

TETRACICLINA **Lederle**

Más rápida absorción

Mínimo de reacciones secundarias

Mayor estabilidad

La **ACROMICINA**, un nuevo antibiótico de amplio espectro que acaba de ser perfeccionado por el Cuerpo Investigador de la Lederle, en el transcurso de pruebas clínicas ha demostrado poseer mayor eficacia, con la ventaja de que se absorbe y difunde con más prontitud por los tejidos y flúidos del organismo, amén de que gracias a su mayor estabilidad redunda en niveles sanguíneos altos y prolongados.

La **ACROMICINA** viene en cápsulas de 250mg, 100mg y 50mg; como polvo S. Ch.* (dispersivo saboreado con chocolate) con 50mg del antibiótico por cucharada (3,0g), y en forma intravenosa con 500mg, 250mg y 100mg por frasco. Con tanta rapidez como la labor de investigación lo permita, se irán ofreciendo nuevas formas terapéuticas. *Marca registrada

Lederle ...*un timbre de honor*

Lederle Laboratories Division Cyanamid Inter-American Corporation
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El eslabón que faltaba
para
Contener las Hemorragias...

ADRENOSEM

Es específico contra las hemorragias capilares postoperatorias y las relacionadas con enfermedades hemorrágicas.

ADRENOSEM es un hemostato sistémico que no interfiere con la presión arterial, la palpitación o el volumen cardíaco.

ADRENOSEM no altera el tiempo de coagulación ni ejerce acción alguna sobre los constituyentes sanguíneo-coagulantes.

ADRENOSEM representa el complejo de adrenocromo monosemicarbazona, el cual es atóxico aún cuando se administra por largo tiempo.

ADRENOSEM produce hemostasis por aumento de la resistencia y disminución de la permeabilidad de los vasos menores y los capilares en casos de:

Tonsilectomía	Hemóptisis
Epistaxis	Cirugía Nasofaríngea
Exudaciones Postoperatorias	Telangiectasia Familiar
Hemorragia Retinal	Cirugía Génitourinaria
Prostatectomía	Púrpura Idiopática

Obtenible en ampollas de 5 mg. y en tabletas de 2.5 mg.

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División de Exportación, 507 West 33rd Street, New York 1, N.Y., U.S.A.

Distribuidores: COMERCIAL GODEL, Calle Europa 802
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Para el Tratamiento de las ANEMIAS

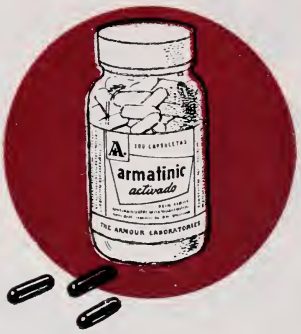
armatinic
activado capsuletas

Cada capsuleta de ARMATINIC ACTIVADO contiene:

Sulfato Ferroso Desecado.....	200 mg.
*Crystamín.....	10 mcg.
Acido Fólico.....	1 mg.
Acido Ascórbico (Vitamina C).....	50 mg.
†Hígado Fracción II, N. F. con Duodeno Desecado.....	350 mg.

*La Vitamina B_{12} Cristalina de los Laboratorios Armour

†El hígado se digiere parcialmente con una cantidad igual de duodeno durante el proceso de manufactura.



PRESENTACIÓN: Frascos de 50 y 100.

Vitamina B_{12} con "activador" más concentraciones efectivas de todos los demás principios hemopoyéticos para asegurar una rápida y completa respuesta hemopoyética.

PRESENTACIÓN: Frascos de 4 y 8 oz.

armatinic
el nuevo LIQUIDO hematínico

Cada onza flúida de ARMATINIC LIQUIDO contiene:

Fracción Hepática I (Clarificada)....	1.25 gm.
Citratos de Hierro y Amonio F.E.U...	1.30 gm.
Acido Fólico.....	2.0 mg.
*Crystamín.....	20.0 mcg.

*La Vitamina B_{12} Cristalina de los Laboratorios Armour

Para una efectiva y rápida respuesta clínica y un sabor refrescante. El Armatinic Líquido suministra Vitamina B_{12} Cristalina con hígado clarificado y otros importantes principios hemopoyéticos en una forma líquida, fácil de tomar, con un sabor agradable.

THE ARMOUR LABORATORIES CHICAGO 11, ILLINOIS, E. U. A.

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TERAPEUTICA FISIOLOGICA MEDIANTE LA INVESTIGACION BIOQUIMICA

This advertisement appears in the November, 1952 issue of the following publications:
America Clinica — El Farmaceutico — Sinopsis Medica Internacional

Distribuidores: LUIS GARRATON, INC.
Fortaleza 352 - Tel. 3-1593 - Apartado 2984 - San Juan, P. R.

NION

INJECTABLE SOLUTIONS

Intramuscular . . . Intravenous

to relieve nausea and vomiting of pregnancy and in adolescent acne

PYRIBEXIN

(Pyridoxine HCl + Thigmine Chloride)

Each 1 cc contains:

Vitamin B1	50 mg.
Vitamin B6	50 mg.

VIALS OF 10 cc

for use in hypochromic and nutritional anemias

IROBLEX

(Iron - Liver - B Complex)

Each cc contains:

Thiamine HCl (B1)	100. mg.
Riboflavin (B2)	0.5 mg.
Pyridoxine HCl (B6)	1. mg.
NICOTINAMIDE	50. mg.
IRON CACODYLATE	10. mg.
LIVER (10 U.S.P. UNITS PER CC)	0.2 cc.

Phenol (As preservative) 0.5%

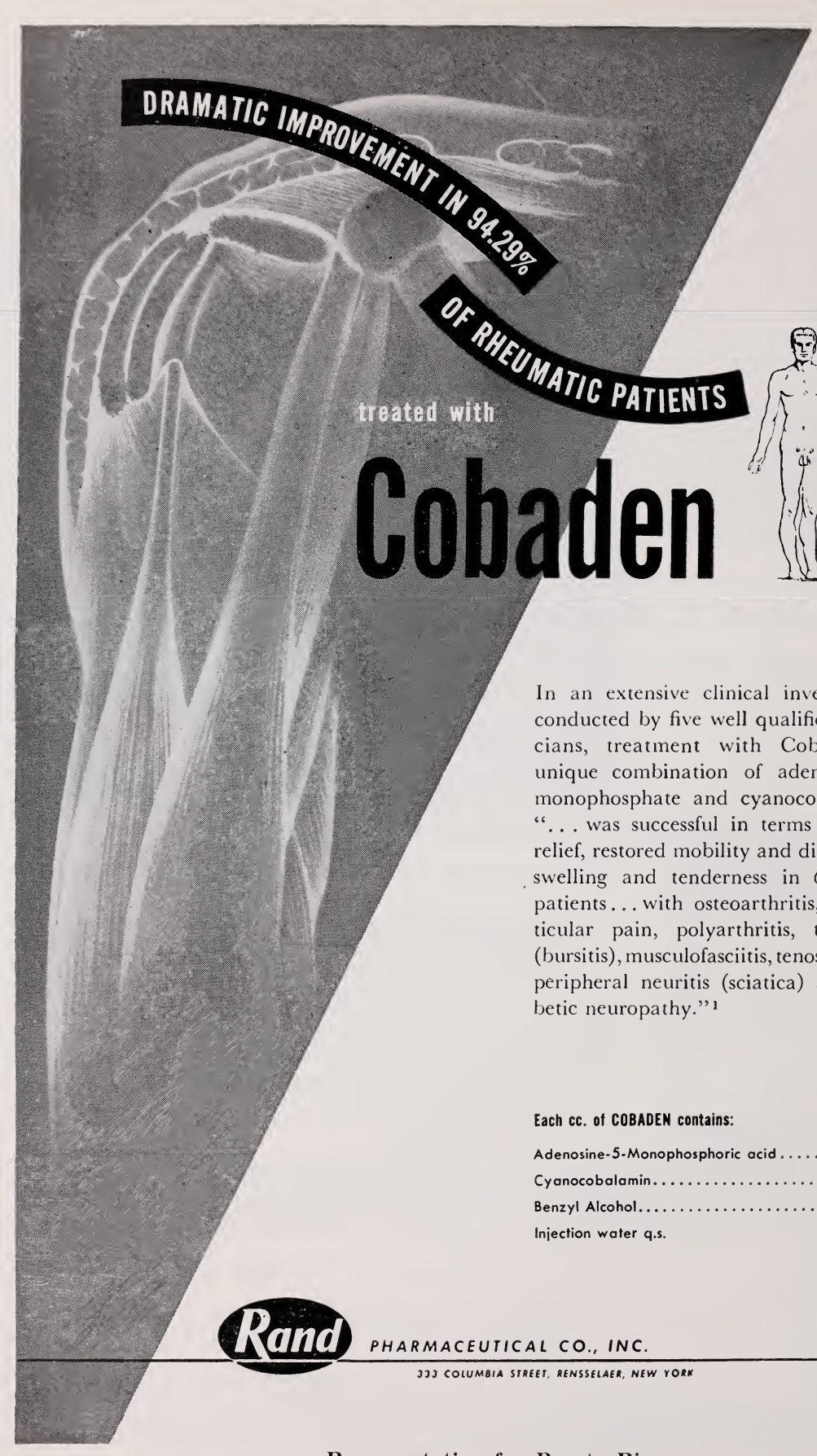
VIALS OF 10 cc

← Improved Formula

NION CORPORATION LOS ANGELES 38, CALIFORNIA

JOAQUIN BELENDEZ SOLA, INC.

P.O. BOX 1188, SAN JUAN, PUERTO RICO

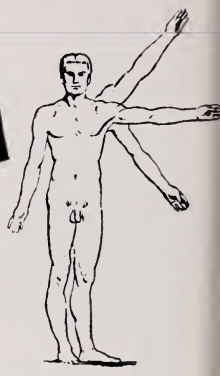


DRAMATIC IMPROVEMENT IN 94.29%

OF RHEUMATIC PATIENTS

treated with

Cobaden



In an extensive clinical investigation conducted by five well qualified physicians, treatment with Cobaden, a unique combination of adenosine-5-monophosphate and cyanocobalamin, "... was successful in terms of pain-relief, restored mobility and diminished swelling and tenderness in 66 of 70 patients... with osteoarthritis, polyarticular pain, polyarthritis, tendinitis (bursitis), musculofasciitis, tenosynovitis, peripheral neuritis (sciatica) and diabetic neuropathy."[1]

Each cc. of COBADEN contains:

Adenosine-5-Monophosphoric acid ..... 25 mg.
Cyanocobalamin ..... 60 mcg.
Benzyl Alcohol 1.5%
Injection water q.s.

Rand PHARMACEUTICAL CO., INC.

333 COLUMBIA STREET, RENSSELAER, NEW YORK

Representative for Puerto Rico
A. F. LEGRAND, Ph.G.
P. O. Box 9022 — Santurce 29, P. R.

UNA MANERA SENCILLA DE ESTIMULAR LA

BUENA NUTRICION

Todos los alimentos BEECH-NUT para bebés han sido aceptados por el Consejo de Alimentos y Nutrición de la Asociación Médica Americana, como también todas las afirmaciones en cada anuncio de Alimentos BEECH-NUT para bebés.

Cuando una madre preocupada le pregunte qué hacer para que su bebé coma más, usted puede ayudarle a comprender que un niño obtiene mayores beneficios de sus alimentos cuando los come con gusto.

No se puede esperar que un niño prospere desde el punto de vista nutricional ni emotivo, cuando las horas de sus comidas están importunadas por ruegos y contrariedades.

Es beneficioso para sus pequeños pacientes el que BEECH-NUT incorpore al agradable sabor de sus alimentos un positivo valor nutritivo.

Ahora, con más variedad que nunca entre la cual escoger, BEECH-NUT hace más fácil que las madres complazcan a los pequeños pacientes de usted, y mantener felices las horas de las comidas.

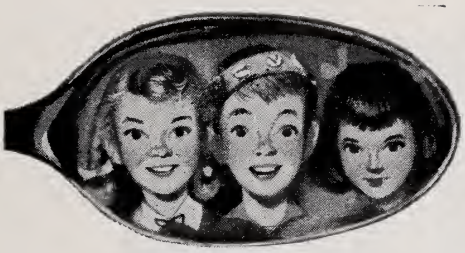
Una amplia variedad que usted puede recomendar: Sopas de Carne y de Vegetales, Frutas, Vegetales y Postres.

BEECH-NUT ALIMENTOS para NIÑOS

Envasados en frascos de cristal

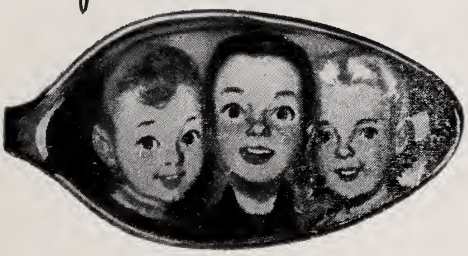
Distribuidores Exclusivos para Puerto Rico:

JOSE MALGOR y CIA., Tetuán 305, San Juan, Puerto Rico—Teléfono: 2-2200



Mulcin

puts a smile in the vitamin spoon



It's the taste of Mulcin that all children like . . . the refreshing flavor of real orange. It's the ready acceptance of Mulcin that all mothers appreciate . . . no more need to coax or bribe even finicky children.

The light, smooth texture of this vitamin emulsion makes pouring easy. And Mulcin needs no refrigeration; even at room temperature its potency is assured.

Each teaspoon of MULCIN supplies:

Vitamin A	3000 Units
Vitamin D	1000 Units
Ascorbic Acid	50 mg.
Thiamine	1 mg.
Riboflavin	1.2 mg.
Niacinamide	8 mg.

Available in 4 oz. and economical 16 oz. bottles.

Mulcin

MEAD

MEAD JOHNSON & COMPANY
Evansville 21, Ind., U.S.A.

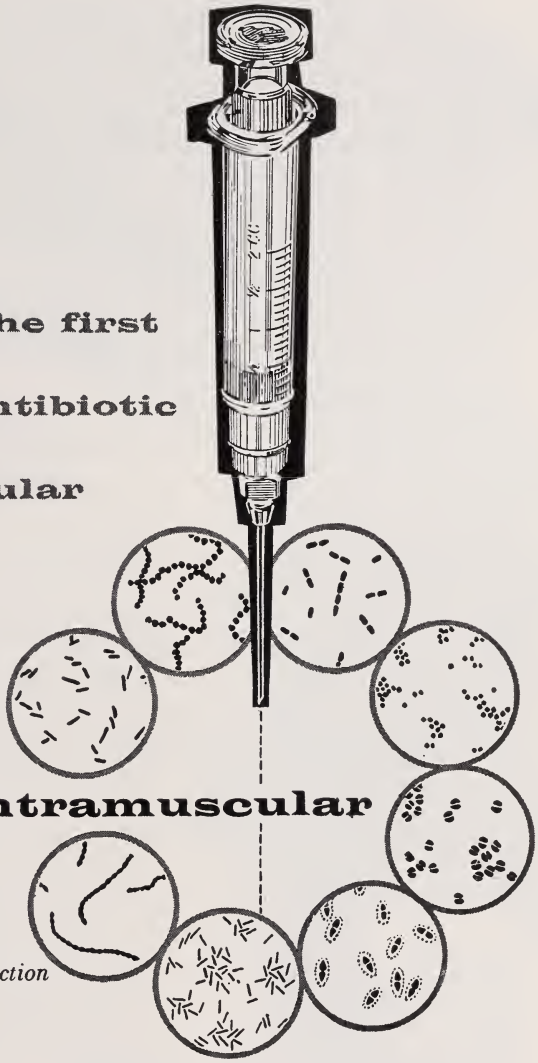
P. O. Box 3081 — San Juan, P. R.

New...the first

broad-spectrum antibiotic

for intramuscular

injection...



Terramycin* Intramuscular

(brand of oxytetracycline)

easy parenteral administration

prompt broad-spectrum antibiotic action

well tolerated

TERRAMYCIN INTRAMUSCULAR is available in one-dose vials containing 100 mg. of sterile TERRAMYCIN Hydrochloride. The dosage is 200 to 400 mg. daily, given in divided doses of 100 mg. each at 6 to 12-hour intervals.

*TRADEMARK OF CHAS. PFIZER & CO., INC.

Sole distributors:

Representing

Pfizer

EST 1849

World's Largest Producer of Antibiotics

JX 807 E PRINTED IN U.S.A.

DUAL-PROCESSED FOR

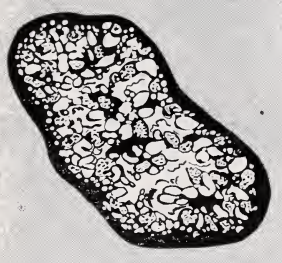
Greater nutrient availability...

...EASIER DIGESTIBILITY
...SMOOTHER TEXTURE

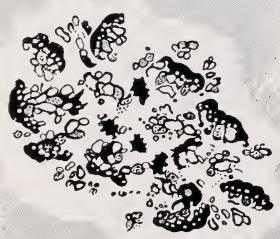
In the preparation of vegetables and fruits as baby foods, home-sieving and commercial straining produce a relatively fine subdivision of the cooked food. Under the microscope, foods so prepared are characterized largely by the predominance of readily identified intact food cells and by long coarse cellulose fibers.

To accomplish a still finer subdivision . . . one more suitable for early supplementation of the infant's milk diet . . . Libby places strained foods through an additional process—homogenization—and thus advantageously changes their physical structure. In this manner, the finest practicable subdivision of the vegetable cell is attained, and the contained nutriment becomes homogeneously dispersed throughout the mixture.

In consequence, Libby's Baby Foods present greater nutrient availability, easier digestibility, smoother texture; they can be bottle-fed as part of the "formula" without perceptibly retarding the rate of flow through a nipple opening of normal size; they may be fed . . . and are well tolerated . . . as early as the fifth week of life. And their cellulose content, comminuted to bland ultra-small particles, maintains an unimpaired "bulk" action.



Intact food cell, characteristic of strained foods. The nutriment is retained within the cellulose fiber capsule as a structural unit.



After homogenization: The capsule is ruptured and comminuted, the nutriment is released—subdivided—and dispersed, exposing a large surface area to the action of digestive juices.

Libby, McNeill & Libby
Chicago 9, Illinois

Beets • Carrots • Green Beans • Peas
Spinach • Vegetable Soup • Mixed Vegetables • Garden Vegetables • Liver Soup • Vegetables with Beef • Apples & Apricots • Apples & Prunes • Apple Sauce • Peaches • Peaches-Pears-Apricots • Prunes (With Pineapple Juice and Lemon Juice) • Custard Pudding

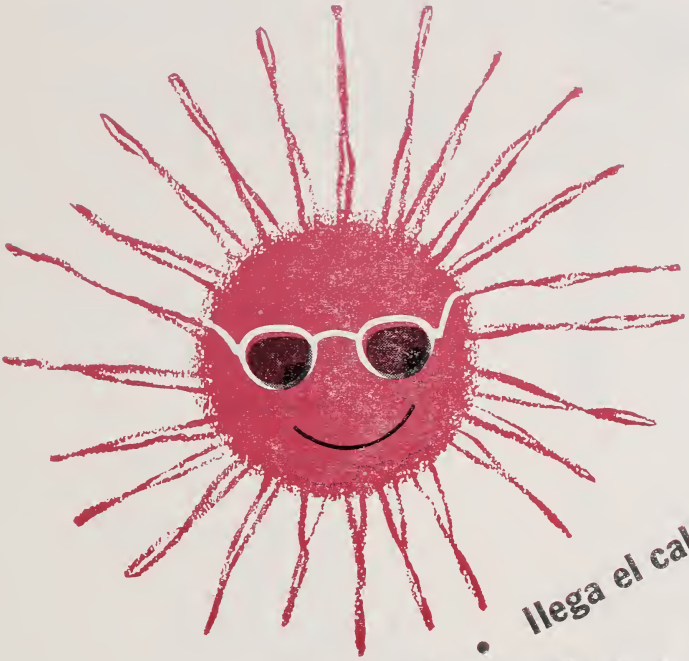
Vegetables with Bacon • Vegetables with Lamb • Apricots-Farina • Pears & Pineapple • Banana Pudding

The last five items not yet submitted to the Council on Foods and Nutrition. All other items Council Accepted.

Libby's

ACCEPTED AMERICAN MEDICAL ASSN. Council on Foods and Nutrition

HOMOGENIZED BABY FOODS



Llega el verano • Llega el calmante

CALADRYL

LOCION Y CREMA
DE CALAMINA Y BENADRYL

El Caladryl proporciona el tan deseado alivio del prurito, ardor y otras molestias producidas por las quemaduras de sol, sudamina, y erupciones debidas a pañales húmedos o cosméticos, picaduras de insectos y varicela. Por contener uno por ciento de Benadryl, ejerce una acción antihistamínica y antipruritica excepcionalmente eficaz. El excipiente de calamina, el alcanfor y la glicerina que contiene el Caladryl proporcionan una acción calmante.

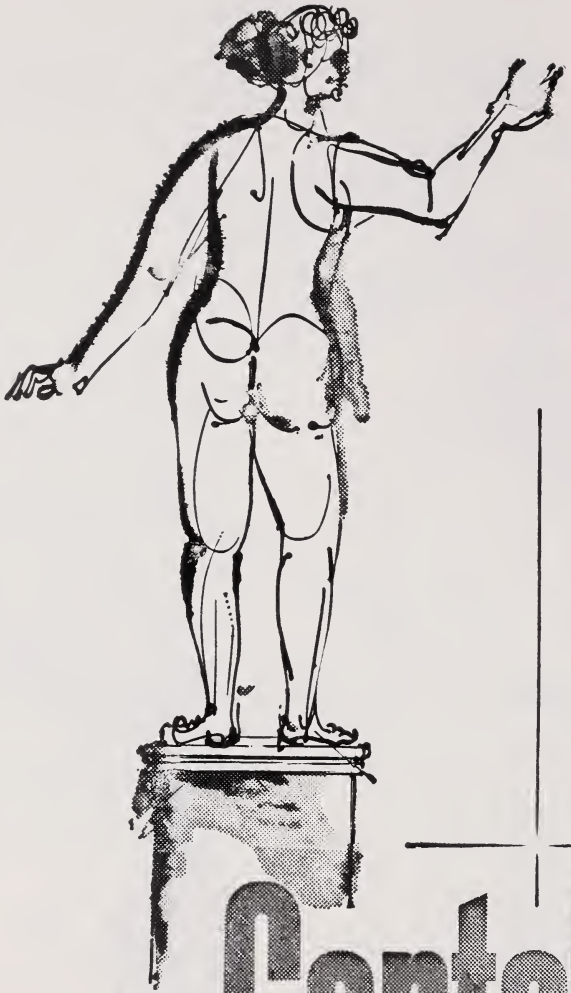
El Caladryl, en forma de loción, se suministra en frascos de 180 cm.³ Se mantiene en estado de suspensión por días—vuelve fácilmente al estado de suspensión con sólo agitar ligeramente.

La Crema de Caladryl se suministra en tubos de 42 Gm. Tiene un excipiente acuomiscible—puede ser aplicada y removida fácilmente.

MEDICAMENTA VERA · P D & CO.

Parke, Davis & Compañía
DETROIT, MICHIGAN, E.U.A.

S-J-11-53



en los procesos dermatologicos comunes

dermatitis seborreica
neurodermatitis
dermatitis por contacto
prurito anal
prurito vulvar
dermatitis atópica
(eczema alérgico)

rápido dominio

con ungüento de acetato de

Cortef*

*marca de la hidrocortisona (compuesto F)

tubos de 5 gm. de ungüento de acetato Cortef en concentraciones de 2.5 mg. (25 mg. por gm.) y 1 % (10 mg. por gm.)

Se remite literatura médica a solicitud.

*MARCA DE FABRICA

Upjohn *Productos farmacéuticos superiores desde 1886.*

THE UPJOHN COMPANY, Kalamazoo, Michigan, U.S.A.

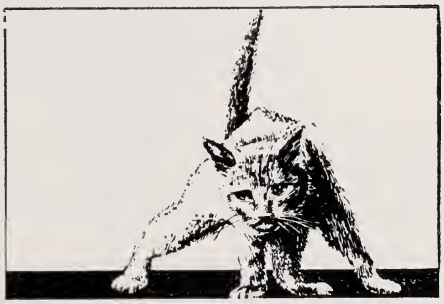
UPJOHN OF ENGLAND, LTD.
4 Aldford Street, Park Lane, London W 1, England.

NEW FORMULA FOR GREATER SAFETY

DICRYSTICIN NEW FORMULA

Squibb Procaine Penicillin G, with
Potassium Penicillin G, plus
Streptomycin Sulfate *and*
Dihydrostreptomycin Sulfate

Ototoxicity is largely eliminated by including *equal parts* of streptomycin and dihydrostreptomycin. Toxicity of streptomycin is chiefly directed against the vestibular branch of the eighth cranial nerve, whereas dihydrostreptomycin is prone to attack the cochlear branch. When the patient gets only half as much of each drug, toxicity is reduced appreciably. Therapeutic effect is undiminished.



Cat treated with streptomycin is ataxic.

Cat treated with the same amount of streptomycin-dihydrostreptomycin has normal equilibrium.

These sketches were made from actual photographs.

Supplied in four potencies to allow for flexibility of dosage

	DICRYSTICIN NEW FORMULA	DICRYSTICIN FORTIS	DICRYSTICIN 800	DICRYSTICIN 800 FORTIS
Units fortified procaine penicillin G	400,000	400,000	800,000	800,000
Grams streptomycin-dihydrostreptomycin	0.5	1	0.5	1

1 dose vials

SQUIBB

leader in the research and manufacture of penicillin and streptomycin

'DICRYSTICIN' is a registered trademark

ANTIBIOSIS MAXIMA

en la boca y la garganta

En las pastillas TYROZETS se han combinado las notables propiedades *antibióticas* y *microbicidas* de la tirotricina, con la conocida acción *analgésica* de la benzocaína.

Resultado: una fórmula terapéutica ideal en forma de pastillas de sabor muy agradable y de gran eficacia medicamentosa.

TYROZETS ataca los gérmenes patógenos en la cavidad bucal y combate la formación de focos infecciosos.

TYROZETS calma rápidamente la irritación y el dolor de garganta.

Rp. **TYROZETS para la asepsia bucal y faríngea, procesos anginosos, en las post-tonsilectomías, etc.**

TYROZETS

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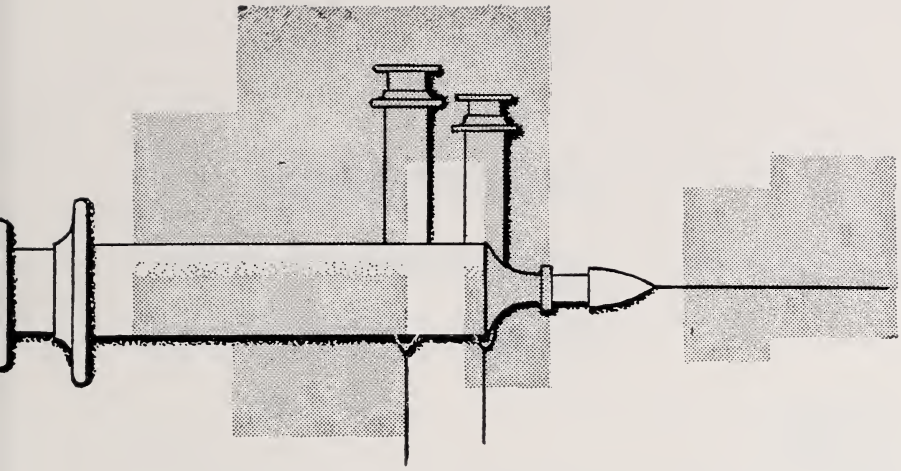

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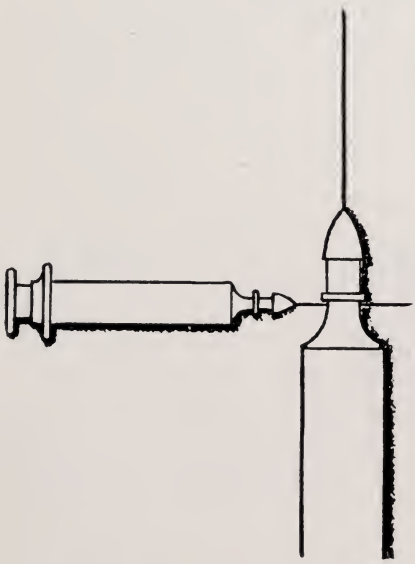

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PRIMER CASO AUTOCTONO DE CISTICERCOSIS EN PUERTO RICO

INFORME DE UN CASO CON HALLAZGOS NECROPSICOS

ENRIQUE KOPPISCH, M.D., F.A.C.P., RAUL MARCIAL ROJAS, M.D., RICARDO CORDERO, M.D., y LUIS GUZMAN LOPEZ, M.D.

San Juan, Puerto Rico

Se informa este caso por ser el primero de esta parasitosis que se ve en Puerto Rico, ya que, por lo demás, es un caso enteramente característico de la forma cerebral del **Cysticercus cellulosae.**

Según Oliver González,[1] hace ya de 14 a 18 años que se tiene conocimiento de la existencia de la cisticercosis en cerdos en la región de Morovis, pueblo del cual era natural, y donde vivió hasta su última enfermedad, la persona cuyo caso aquí informamos.

Fase evolutiva definitiva con el parásito adulto en intestino:

La **Taenia solium** adulta se desarrolla en el intestino delgado del hombre, el huésped definitivo, cuando éste come carne de cerdo, cruda o mal cocida, que contiene los quistes larvarios (**Cysticercus cellulosae**). Al digerirse los quistes, la parte que representa el escólex, o cabeza de la tenia, se desinvagina de su posición natural, y se fija a la mucosa del intestino delgado, creciendo luego, a partir de la cabeza, la solitaria adulta, en el término de dos a tres meses.

El parásito adulto contiene usualmente unos 800 a 1000 segmentos o anillos, y su longitud es de 2 a 4 metros. La cabeza es pequeñísima, de un milímetro de diámetro, y posee una protuberancia retráctil, el rostellum, armada de 26 a 32 ganchos dispuestos en doble círculo. Para la fijación a la mucosa además del rostellum, situado en la parte más alta de la cabeza, existen cuatro

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De los Servicios y Departamentos de Anatomía Patológica y Neurocirugía del Hospital de la Capital, y de la Escuela de Medicina, Escuela de Medicina Tropical, de la Universidad de Puerto Rico.

ventosas laterales. A la cabeza le sigue un **cuello** casi filiforme, de diámetro menor que el de aquélla, y que es liso y relativamente corto (5 a 10 mms.) El resto del cuerpo se denomina **estróbilo,** y se compone de **segmentos, proglótides** o **anillos.** Algunos autores usan el término "estróbilo" para designar la tenia adulta completa.

Los primeros anillos son pequeños, cortos, y más anchos que largos. Su longitud aumenta progresivamente, de modo que hacia el tercio medio del parásito son de forma cuadrada, y más distalmente se tornan más largos que anchos. Hacia el final del estróbilo alcanzan una longitud de 1 a 2 cms., mientras que su anchura es de 0.5 cm. En el parásito vivo los segmentos cambian contínuamente de forma y tamaño por movimientos de contracción que les ensanchan y acortan, y de relajación, que les alargan y estrechan.

Los anillos poseen un útero, ovarios y testículos. Las ovas fecundadas por los espermatozoarios van acumulándose en el útero. Este último es estrecho y alargado en los anillos ingrávidos de las porciones proximales del estróbilo. En las porciones más distales o terminales, al cargarse de ovas fecundadas, el útero se distiende y forma unas ramificaciones laterales que se disponen perpendicularmente al eje longitudinal.

El número de ramificaciones laterales del útero es uno de los distintivos más importantes para la identificación del parásito, pues en el caso de la **T. solium** fluctúa entre 5 y 10, a cada lado de la línea media, mientras que en la **T. saginata** es de 15 a 30.

Los anillos grávidos van desprendiéndose de las porciones distales del estróbilo, aisladamente o en cadena. Cuando esto sucede, se desgarran las ramificaciones uterinas laterales, pasando así los huevos a la luz intestinal.

El huevo de **T. solium** mide de 30 a 40 micras de largo por 20 a 30 de ancho, y se compone de una gruesa cubierta (el embrióforo) de color pardo, finamente radiada, refringente hacia el interior y opaca hacia el exterior. Dentro del embrióforo se encuentra el embrión hexacanto, que así se llama por estar provisto de seis ganchos.

El diagnóstico coprológico tiene que efectuarse por la diferenciación de los anillos expulsados con las heces, ya que los huevos de **T. solium** y **T. saginata** son idénticos. Si se expulsa el verme entero con su escólex, se encontrará que en el caso de **T. solium** éste es globuloso, con un rostellum armado de ganchos en doble hilera. El escólex de **T. saginata** es algo mayor, cuadrangular, y sin rostellum ni ganchos. La mayor longitud de **T. saginata,** y ciertas diferencias en la disposición de los poros genitales y en la conformación de la vagina, son factores adicionales para la identificación de la especie de estos parásitos.

La sintomatología inducida por la presencia en el intestino del

verme adulto es nula o escasa. Cuando aparecen síntomas, estos generalmente se manifiestan como vagos trastornos de la digestión. En algunos niños sobrevienen períodos de aumento del apetito, a pesar de lo cual pueden aquejar debilidad, pérdida de peso, y mareos. Se han descrito numerosos síntomas de origen nervioso, entre éstos el insomnio y, a veces, convulsiones de diversos tipos.[2] Raras veces, la penetración de segmentos en la luz apendicular puede provocar un cuadro de apendicitis aguda. Según Kourí y Basnuevo,[3] en sus casos de esta índole, con operación quirúrgica de urgencia, no se vieron alteraciones del órgano, ni macro-ni microscópicas. Los libros de texto han recogido rarísimos casos en que la cabeza de la tenia (tanto **solium** como **saginata**) ha perforado la pared intestinal, produciendo peritonitis.

A menudo, los movimientos de reptación de los anillos les permite salir por el ano al exterior, con el consiguiente efecto psíquico, muy mortificante y deplorable, en el enfermo. Esa eventualidad, sin embargo, puede aprovecharla el médico, no sólo para establecer con certeza el diagnóstico de teniasis, sino que también para examinar los anillos entre dos láminas de cristal, y comprobar la especie a que pertenecen. La tenia adulta puede vivir en el intestino durante muchos años, habiéndose informado casos, según Belding,[4] de hasta 25 años de infestación.

Fase evolutiva intermedia de quistes larvarios en tejidos (cisticercosis):

El hombre es el huésped definitivo de la **T. solium**, siendo el cerdo el intermediario. Otros animales que pueden servir de huésped intermediario son el jabalí, la oveja, los venados, perros y gatos, así como los monos antropoides. El cerdo, animal coprófago, se infecta al ingerir excremento humano, y como el excremento a menudo contiene anillos sueltos o en cadenas, recargado cada uno de ellos de miles de huevos, la infestación en este animal a menudo es masiva.

La digestión del embrióforo libera el embrión hexacanto u oncosfera, el cual penetra directamente la pared del intestino delgado, alcanzando la luz de vasos sanguíneos o linfáticos. En esta forma son transportados los embriones a diversos órganos y tejidos, pero la localización más frecuente en el cerdo es el músculo esquelético, sobre todo en la lengua, cuello y hombros. Otros músculos, el corazón, hígado, cerebro, pulmones y ojos pueden albergar quistes.

En los tejidos la larva forma un quiste traslúcido u opalino, de delgadísima pared, con un punto blanquecino y opaco que representa el escólex invaginado. Estos quistes o vesículas son ovoides, y alcanzan su tamaño máximo, de 10 por 5 mms., hacia la novena o décima semana.

El hombre puede adquirir la cisticercosis en una de tres formas: (1ª) Al ingerir huevos en el alimento o bebidas contaminadas por heces fecales; (2ª) llevándose huevos a la boca con las manos contaminadas, en casos de portadores de tenia, y (3ª) se ha pensado que en algunos casos sobreviene la autoinfestación cuando los huevos son transportados al estómago por intermedio de movimientos peristálticos retrógrados.

Como quiera que esto suceda, pasa entonces lo mismo que en el huésped intermediario usual (el cerdo): se digieren los embrióforos, liberándose el embrión, el cual penetra en la pared intestinal, y va a situarse en diversos órganos y tejidos. En el hombre la localización más frecuente es en el tejido subcutáneo, seguido en frecuencia decreciente, por el cerebro, ojos, músculos del esqueleto, corazón, hígado, pulmón y peritoneo.

Su aspecto y tamaño son como en el cerdo, excepto que en el cerebro su diámetro máximo puede ser de 2 cms., siendo lo usual 1 cm. Donde los tejidos adyacentes comprimen las vesículas, en músculos, por ejemplo, éstas tienden a ser más pequeñas, algo alargadas y más estrechas que en superficies relativamente libres, como las meninges, el espéndimo ventricular y la retina.

El embrión, al alcanzar los tejidos, produce una leve infiltración linfocitaria, y la formación de una delgada cápsula fibrosa, dentro de la cual está situado el escólex invaginado. Si muere la larva, primero sobreviene un aumento en el líquido, que luego va espesándose, el quiste se contrae, se torna opaco y semisólido, y surge alrededor de él una reacción inflamatoria más notable que cuando sobrevive la larva. Se caracteriza esta reacción por una densa infiltración de polinucleares, linfocitos, células plasmáticas, células epitelioideas y macrófagos vacuolados, y por la formación de algunas células gigantes del tipo de reacción a cuerpo extraño. En el cerebro y meninges se desarrolla una endarteritis obliterante en los vasos en esa zona y aún a alguna distancia de ella. La reacción exudativa es gradualmente reemplazada por proliferación fibroblástica. En algunas situaciones, como en músculos, es frecuente la calcificación final.

La sintomatología de la fase larvaria está sujeta, naturalmente, a dos factores principales, que son la localización de los quistes y su número.

Los quistes subcutáneos son de gran importancia diagnóstica. Estos pueden ser ligeramente visibles, o se les puede palpar, generalmente en el torso, cuello, cara y extremidades, como nódulos firmes, redondeados u ovoides. En esta situación puede que algunos no sean palpables hasta que muera la larva, con el consiguiente aumento en el líquido del quiste. Como luego va absorbiéndose este líquido, y al mismo tiempo otros nódulos van hacién-

dose palpables, en algunos casos se obtiene la impresión de que los nódulos son evanescentes o migratorios.

En el ojo por lo general sólo se forma un quiste en una de dos posiciones: (a) extraocularmente, por debajo de la conjuntiva del canto interno o, (b) dentro del globo ocular. En esta segunda posición el quiste se desarrolla por debajo de la retina, haciendo proyección hacia la cámara vítrea. Esto generalmente provoca una iridociclitis, a veces con oclusión pupilar.

En el cerebro los quistes son más frecuentes en las leptomeninges, pero pueden también asentarse en la sustancia cerebral o en el revestimiento ependimario de los ventrículos. Son más frecuentes en las leptomeninges de las porciones basales y a lo largo de las cisuras temporoparietales. A veces los quistes se agrupan como racimos de uvas, disposición que se denomina **cisticerco racemoso.** El líquido cefalorraquídeo muestra aumento en su presión, en globulina y en linfocitos, y a veces contiene eosinófilos.

Las manifestaciones clínicas son muy variables. Valladares y sus colaboradores[5] agrupan los casos de cisticercosis cerebral en la siguiente forma.

A— Clasificación sindrómica de las formas cerebrales.

1— Los casos que se caracterizan por hipertensión intracraneana, con leves alteraciones mentales y signos neurológicos ausentes o escasos. Prácticamente, el cuadro clínico se compone de cefalea, vómitos en proyectil y edema papilar, discretos trastornos de la memoria con desorientación témporo-espacial, y disturbios cerebelosos o de algún nervio craneano.

2— Los casos en que se añaden, al cuadro de hipertensión intracraneana, numerosos signos neurológicos de distribución difusa que hacen difícil un diagnóstico topográfico: diplopia, nistagmo, alteraciones reflejas y sensitivas del trigémino, paresia facial central, acúfenos, sordera, paresia lingual, ataxia, parestesias en extremidades, signos piramidales, etc. Los trastornos mentales pueden ser más acusados, y puede haber confusión mental.

3— Los casos en que los signos de hipertensión intracraneana van unidos a signos focales sugestivos de la presencia de una neoplasia. La epilepsia jacksoniana es una de las expresiones clínicas más importantes.

4— Los casos en que las manifestaciones clínicas son predominante o exclusivamente mentales. Puede haber signos de hipertensión intracraneana, pero la sintomatología es esencialmente demencial, casi siempre de tipo esquizofrénico.

B— Formas de localización cerebral.

1— Cisticercosis uniquística: En esta forma sólo se encuentra un quiste cisticercósico solitario. Su localización preferente es

en el orden siguiente: (a) En el cuarto ventrículo, (b) en la sustancia cerebral, y (c) en la corteza cerebral. Generalmente corresponde esta localización a los cuadros sindrómicos 1 y 3, y el pronóstico operatorio suele ser bueno.

2— Cisticercosis multiquística: Es típica de esta variedad la localización en la sustancia cerebral, pero también pueden situarse los quistes en las leptomeninges, como en nuestro caso, y además en los ventrículos. Esta variedad topográfica corresponde a menudo con las formas sindrómicas 2 y 4.

3— Cisticercosis racemosa: Como lo indica la designación, los quistes se agrupan en racimos. A menudo son de ubicación cisternal, pero pueden tener otras localizaciones. Más frecuentemente corresponden con las formas sindrómicas 2 y 4.

4— Cisticercosis generalizada: En esta forma, que es relativamente rara, pueden presentarse las más diversas formas de localización, dando al cuadro un mayor polimorfismo clínico.

MacArthur[6] le ha dado énfasis a la importancia de la epilepsia de comienzo tardío como manifestación de una cisticercosis cerebral, según sus estudios en soldados británicos estacionados en la India y en Egipto.

La localización en músculos esqueléticos suele ser silenciosa, desde el punto de vista clínico, pero el enfermo a veces se percata de la presencia de nódulos en los músculos o en el tejido subcutáneo. En raros casos el período invasivo se acompaña de reacción febril y síntomas generales. La localización muscular puede revelarse radiográficamente, años más tarde, como pequeñas calcificaciones bien definidas, más largas que anchas.

Diagnóstico de la cisticercosis:

El diagnóstico de la forma larvaria suele ser difícil. Lo que más facilita el diagnóstico es la presencia de nódulos subcutáneos o en músculos. La calcificación de los quistes generalmente no ocurre hasta los cuatro o cinco años de adquirida la infestación. Casi nunca se pensará en la posibilidad de esta parasitosis a menos que se tenga conocimiento de la existencia del **Cysticercus cellulosae** en la región. El comienzo de ataques epilépticos en adultos sin antecedentes debe siempre de hacer pensar en la posibilidad de esta parasitosis. La prueba de la fijación del complemento es útil, pero su negatividad no excluye el diagnóstico.

INFORME DEL CASO

Enferma F. R. Q. Autopsia No. 2104. Historial clínico No. 70633. Edad 39 años; raza blanca. Durante los dos últimos años

ha estado sufriendo de frecuentes e intensas cefaleas que se aliviaban con el uso de analgésicos corrientes hasta cinco semanas atrás. En el último mes han sido casi constantes y de gran intensidad. Se prolongan todo el día y la noche y le interrumpen el sueño. Son generalizadas, pero de mayor intensidad en la región frontal. Se acompañan de náuseas y vómitos de tipo cerebral. Ha tenido dolores de cabeza muy intensos, durante los cuales se le ha nublado la vista y ha caído al suelo. Estos episodios han durado de 5 a 10 minutos, sintiéndose luego mejor. En los últimos tres días le ha aparecido una desviación de la comisura bucal hacia el lado derecho y adormecimiento y hormigueo de la mano y pierna del lado izquierdo.

Sus antecedentes patológicos personales y familiares no son significativos.

Examen físico: Temperatura 98.2ºF. Presión arterial 110/70. Pulso 80. Respiraciones 21. Desarrollo físico y estado nutritivo normales.

Examen neurológico: Pupilas isocóricas de tamaño normal; reaccionan a la luz y a la acomodación. Edema papilar bilateral de tres dioptrías. En el ojo izquierdo se encuentran hemorragias peripapilares recientes. Paresia facial central izquierda. Discreta paresia de la extremidad inferior izquierda. Reflejos abdominales ligeramente más activos en el lado derecho.

Exámenes de laboratorio: Hematíes 4,760,000. Leucocitos 7,400. Polimorfonucleares 51%. Linfocitos 49%. Hemoglobina 94%. Orina: Normal.

Roentgenología: No presenta signos de hipertensión intracraneal, ni de alteraciones óseas.

Diagnóstico clínico: Tumor cerebral frontal derecho, parasagital, probablemente un astrocitoma.

Ventriculografía: Trepanaciones occipito-parietales bajo anestesia local con novocaína al 1%. Sistema ventricular simétrico. Discreta hidrocefalia. El acueducto de Silvio y el cuarto ventrículo no se visualizan. La imagen del tercer ventrículo no está claramente definida. Interpretación: Tumor del tercer ventrículo.

Operación: Se llevó a cabo inmediatamente después de la ventriculografía, el día 18 de setiembre de 1950, con anestesia endotraqueal por óxido nitroso, oxígeno y pentotal sódico. Se practicó una incisión longitudinal en la línea media occipito-cervical, con resección de la escama occipital y del arco posterior del atlas, y con apertura de la duramadre, exponiendo la fosa posterior. La exploración de las estructuras nerviosas fué prácticamente imposible debido al intenso edema cerebral. Al rechazar hacia afuera los hemisferios cerebelosos para poner al descubierto el cuarto ventrículo, el tejido nervioso se desgarraba con facilidad y sangraba profusa-

mente. Mientras tanto, la función respiratoria se hacía cada vez más deficiente. Tomando en consideración estas condiciones de edema cerebral, hemorragia y déficit respiratorio, se decidió terminar el acto quirúrgico. Sólo fué posible cerrar la piel, ya que el edema impedía aproximar los planos musculares sin destruir los hemisferios cerebelosos al atar los hilos de sutura. La enferma falleció una hora y 45 minutos después de la operación, en fallo respiratorio.

INFORME DE AUTOPSIA

Descripción macroscópica:

Cadáver de una mujer de 39 años de edad, de la raza blanca, en buen estado de desarrollo y nutrición.

Cavidades abdominal y torácica: Normales.

Cavidad craneana: Los detalles quirúrgicos aparecen con la descripción clínica. Se encontró escasa cantidad de sangre líquida y coagulada en el espacio subdural, en región occipital.

Cerebro: Pesó 1180 gmos. Las circunvoluciones cerebrales estaban ensanchadas y algo aplanadas. La sustancia cerebral se herniaba por los orificios de trepanación. Por lo que respecta a la cisticercosis los hallazgos fueron los siguientes. Los ventrículos aparecían ligeramente dilatados y contenían líquido sanguinolento. Se encontraron seis quistes, con el aspecto y localización siguientes:

1—En la leptomeninge correspondiente al lóbulo temporal e ínsula de Reil del lado derecho. Este medía 0.5 cm. de diámetro y era redondo, con pared de un espesor de 2 mms. y aspecto interior liso y blanco.

2—En la leptomeninge, por debajo del quiasma óptico, en todo similar al primero.

3—En la leptomeninge, inmediatamente posterior al segundo, y del mismo tamaño y aspecto.

4—En la leptomeninge, por debajo de los cuerpos mamilares, con diámetro de 1 cm., y parecido a los anteriores.

5—Nódulo redondeado, pardo-grisáceo, de 0.5 cm. de diámetro, y situado inmediatamente por delante del quiste No. 4.

6—Nódulo sólido, de 0.3 cm., blanco-grisáceo, situado en el espacio subaracnoideo a la izquierda del bulbo raquídeo.

En múltiples cortes del encéfalo y del cerebelo no se encontró ningún quiste, aparte de los seis reseñados.

Se examinaron todas las vísceras, encontrándose alteraciones patológicas sólo en las siguientes:

Aorta: Depósitos ateromatosos mínimos en la íntima.

Pulmones: Ambos estaban distendidos por enfisema y edema.

Al corte eran muy congestivos y los tejidos manaban líquido espumoso y algo sanguinolento.

Vejiga urinaria: Aparecía distendida por 1500 cc. de orina turbia.

Utero: El endometrio era de aspecto hemorrágico.

Ovario izquierdo: Contenía un cuerpo amarillo en regresión.

Ovario derecho: Pequeño quiste luteínico de 1.5 cms. de diámetro.

Intestino grueso: El ciego mostraba un pólipo pediculado de 0.5 cm. de diámetro.

Tiroides: En el lóbulo derecho apareció un nódulo amarillento y firme de 0.5 cm. de diámetro.

Descripción microscópica:

Pulmones: Todos los vasos sanguíneos aparecían muy congestionados. Los espacios alveolares estaban ocupados por proteína coagulada, eritrocitos y macrófagos.

Bazo: Era de notar la escasez de linfocitos en los folículos de Malpigio.

Páncreas: Se notaba fibrosis de la íntima de las arteriolas.

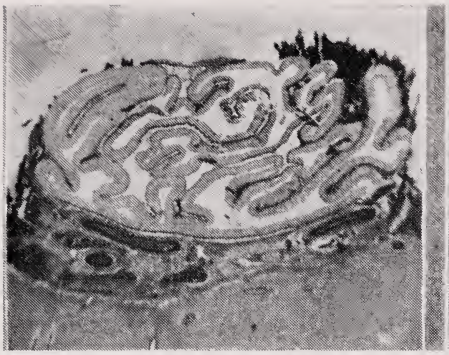
Utero: El endometrio estaba en fase menstrual.

Ovarios: En uno de ellos se vió un cuerpo amarillo en estado de madurez, pero con centro hemorrágico.

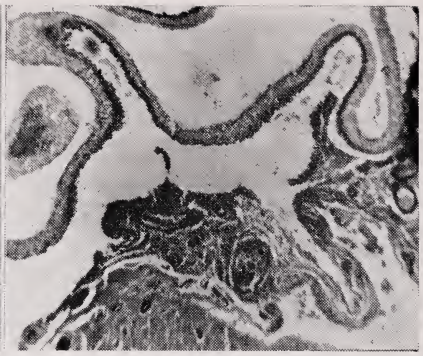
Tiroides: Un corte era normal. En otro corte se vió un nódulo neoplásico de periferia mal definida. Se componía de células de tipo epitelial que formaban estructuras glandulares y pequeñas proyecciones papilares. Los núcleos eran bastante regulares en forma y tamaño, pero muchos de ellos eran hipercromáticos. Se veían figuras mitóticas poco numerosas. El tumor estaba incompletamente rodeado de una cápsula fibrosa, la cual mostraba invasión por el tejido neoplásico. En algunas partes de la periferia del tumor se encontraban numerosos linfocitos.

Cerebro: Casi todos los grandes quistes estaban bien conservados. (Grabados 1, 2, 3 y 4). Las leptomeninges a su alrededor mostraban ligera infiltración con linfocitos y algunas células linfáticas (Grabados 1, 2 y 3) En algunos campos se advertía una ligera fibrosis. Ninguno de los cortes incluyó el escólex.

El nódulo situado cerca de los cuerpos mamilares (Grabado 1) y el que se hallaba a la izquierda del bulbo raquídeo, estaban completamente colapsados y no mostraban cavidad quística. A pesar de que no se advertían alteraciones en los núcleos de la larva, (Grabados 2 y 3) ambos estaban rodeados de una extensa zona de infiltración con linfocitos y células plasmáticas. Se encontraron campos de necrosis de coagulación, (Grabado 5) y se vieron algunas células gigantes del tipo de reacción a cuerpo extraño. Varias arterias de mediano calibre, situadas en las zonas



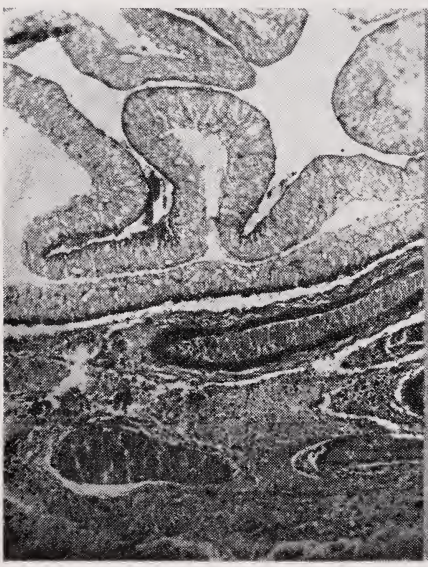
Grabado 1



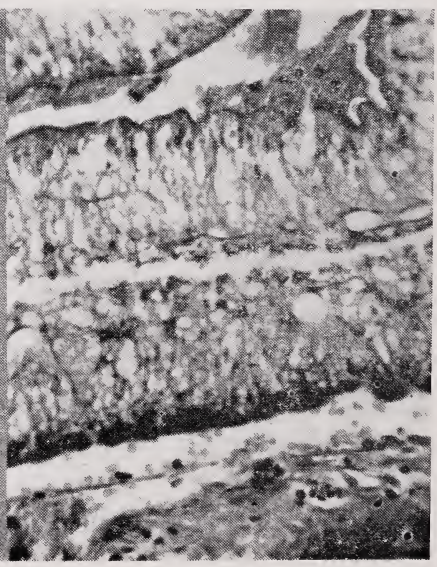
Grabado 2

Grabado 1: Quiste colapsado pero bien conservado; escasa reacción meníngea ---------- 8 X

Grabado 2: Quiste con membrana bien conservada y moderada infiltración linfo-plasmocitaria en leptomeninges ---------- 8 X



Grabado 3



Grabado 4

Grabado 3: Detalle de membrana parasitaria, con edema e infiltración linfocitaria de las leptomeninges ---------- 80 X

Grabado 4: Detalle de membrana parasitaria con sus capas cuticular, germinativa y parenquimatosa ---------- 360 X

de inflamación, aparecían con la íntima bastante engrosada por fibrosis (Grabados 6 y 7). El tejido nervioso subyacente era edematoso, y muchas de las neuronas exhibían alteraciones degenerativas. El edema del tejido nervioso era evidente en diversos cortes a distintos niveles del encéfalo.

Diagnóstico anatómico: Cisticercosis de leptomeninges en región de quiasma óptico, ínsula de Reil derecha, cuerpos mamilares, y bulbo raquídeo, lado izquierdo; operación quirúrgica: craneotomía, ventriculografía y exploración del cerebro; hemorragias cerebrales múltiples, postoperatorias; congestión y edema pulmonar; adenocarcinoma papilar de glándula tiroides; endometrio en fase menstrual; quiste folicular del ovario derecho; gastritis crónica; pólipo del ciego.

COMENTARIOS

Como hasta ahora no se había constatado la existencia de casos de cisticercosis en Puerto Rico, no se pensó en esa parasitosis al estudiar clínicamente este caso. Además, los síntomas y signos eran de neoplasia intracraneana, y se juzgó necesaria una intervención quirúrgica de urgencia.

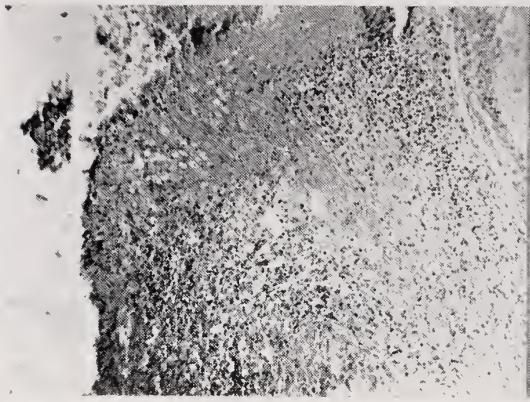
La autopsia reveló un caso de cisticercosis con localización exclusivamente intracraneana, en forma meníngea. La descripción patológica no menciona el escólex en ningún quiste, y tampoco se le encontró en los cortes microscópicos. A pesar de esto no creemos que el diagnóstico pueda ser otro que el señalado.

RESUMEN

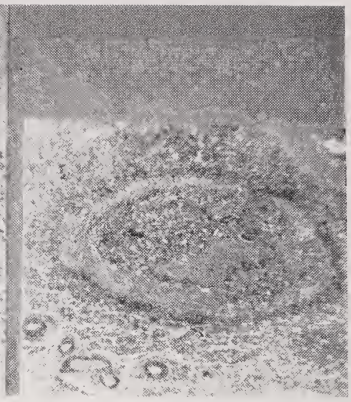
Se informa el primer caso de cisticercosis en Puerto Rico, de asiento cerebral, y con signos de neoplasia intracraneana. La intervención quirúrgica fué infructuosa, y el diagnóstico se estableció en la autopsia. Encontráronse seis quistes, todos en la leptomeninges de las porciones basales, con la excepción de uno situado entre el lóbulo temporal derecho y la ínsula de Reil, y otro situado yuxta-bulbarmente. La enferma provenía de Morovis, región donde se sabe, desde hace algunos años, que existe la cisticercosis entre los cerdos.

SUMMARY

The first autochtonous case of cysticercosis in Puerto Rico is reported. It was of cerebral type, with clinical manifestations of an intracraneal tumor. Death took place shortly after an exploratory craneotomy, and the diagnosis was established at autopsy.



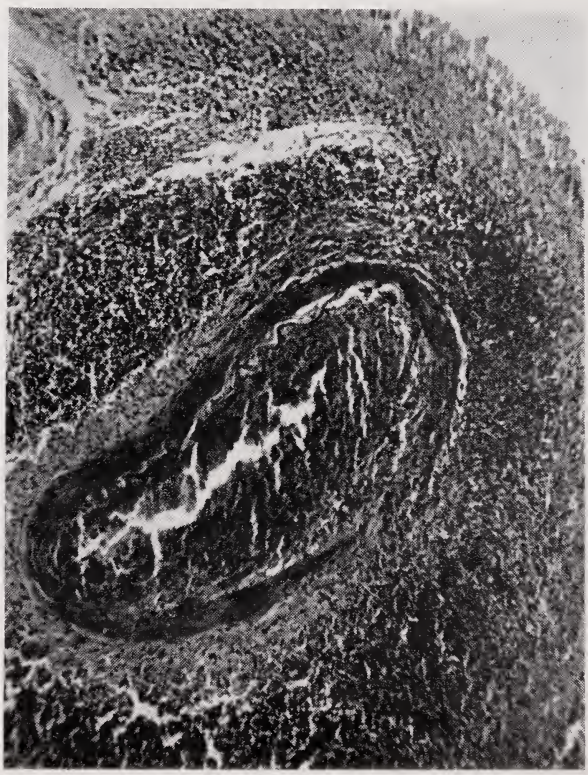
Grabado 5



Grabado 6

Grabado 5: Reacción en torno de quiste con larva muerta. Extensa zona de necrosis de coagulación rodeada de linfocitos ---------- 80 X

Grabado 6: Inflamación de arteria, endarteritis proliferativa y densa infiltración linfocitaria en cercanía de un quiste con larva muerta ---------- 80 X



Grabado 7: Parecida al No. 6 ---------- 8 X

Six cysts were found, all of them basally situated, in the leptomeninges, with the exception of one, which was found between the right temporal lobe and the insula of Reil, and another situated in the leptomeninges to the left of the medulla oblongata. The patient came from Morovis, a region where it has been known for several years that cysticercosis is found among hogs.

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PUNTOS PRACTICOS EN EL TRATAMIENTO DEL PACIENTE DIABETICO*

AGUSTIN M. DE ANDINO, Jr., M.D.**

Una vez el médico ha establecido con certeza el diagnóstico de diabetes mellitus se instituye el tratamiento que combina tres medidas muy importantes, a saber, la dieta, el ejercicio regulado y la insulinoterapia. Los objetivos a alcanzar en el tratamiento de la enfermedad son los siguientes: 1) la nutrición normal del paciente, 2) la restauración del paciente a sus ocupaciones usuales, 3) la prevención de las complicaciones, especialmente de tipo vascular, y 4) la prevención de la hiperglucemia, la glucosuria, la hiperlipemia y la hipercolesterinemia. Debemos señalar también que es muy importante el aleccionar debidamente al paciente a fin de asegurar su cooperación inteligente, lo cual constituye una parte muy importante del tratamiento adecuando de la enfermedad.

Medidas dietéticas

Antes de 1914 los enfermos diabéticos eran alimentados liberalmente, aunque las ventajas de la limitación de la ingestión alimenticia habían sido ya sugeridas y en ocasiones practicadas. La demostración hecha por Allen de que por medio de la sobrealimentación podía provocarse la degeneración de los islotes que quedaban en un perro parcialmente pancreatectomizado, indicaba ya el peligro potencial de tal procedimiento. Siguió entonces una era en que la limitación diabética con sus días de ayuno semanales se convirtió en el medio principal del tratamiento de la diabetes. Con el descubrimiento de la insulina por Banting y Best en 1921 esto ya no fué necesario, y en la actualidad más bien que una limitación se efectúa una regulación de la dieta, lo cual tiene una importancia indudable en el tratamiento de la afección.

En la diabetes muy leve la simple abstención de alimentos dulces y de la azúcar o si ello fracasa, la limitación de los hidrocarbonados de la dieta entre 120 y 150 gramos diarios, puede ser suficiente para suprimir la glucosuria. En tales enfermos no es necesario la administración de la insulina. Este grupo en Puerto Rico constituye alrededor de más del 50% de los pacientes diabéticos encontrados en la práctica corriente. No obstante la presencia de una diabetes leve, este grupo debe quedar sometido a vi-

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* Charla dictada en la reunión anual de la Asociación Médica del Distrito Oeste celebrada en San Germán, Puerto Rico, el domingo 14 de marzo de 1954.

** Profesor Auxiliar de Medicina Clínica, Escuela de Medicina, Universidad de Puerto Rico.

gilancia médica a fin de poner en evidencia cualquier empeoramiento de su estado pues la tendencia es a no seguir el régimen prescrito por el médico.

Cuando la simple restricción dietética que hemos esbozado no es suficiente, se hace necesario un tratamiento dietético más enérgico y posiblemente el empleo de la insulinoterapia. La naturaleza de la dieta del diabético necesita ser considerada desde los siguientes puntos de vista: 1) su valor calórico total, y 2) las proporciones relativas de su contenido en grasas, hidratos de carbono y proteínas. El valor calórico total de la dieta se calcula sumando el requerimiento calórico basal y el factor de actividad. El requerimiento calórico basal se obtiene multiplicando el peso ideal del individuo por 10, a saber, 10 calorías por libra de peso ideal. El factor de actividad representa un porcentaje del requerimiento basal y depende de la ocupación y el grado de actividad del paciente. El factor varía entre un treinta por ciento para una ama de casa o un oficinista y un ochenta por ciento para un jugador de pelota. Algunas autoridades en el campo de la diabetología consideran que es deseable cierto grado de hiponutrición en el diabético y por ello recomiendan que la dieta deba tener un valor calórico total entre un cinco o un diez por ciento por debajo del óptimo normal calculado. Este criterio se basa en el hecho conocido de que la obesidad agrava la enfermedad debido a la relativa resistencia a la insulina que se observa en estos sujetos.

Nosotros consideramos poco deseable el mantener al diabético sometido a una dieta de valor calórico subnormal, ya que éste requiere una dieta normal tanto en cantidad como en calidad. La única excepción a esto se encuentra en el tratamiento del paciente obeso en el cual es imperativo la reducción del peso al nivel normal lo más rápidamente posible. En el paciente que requiera insulina y cuyo peso está entre los límites normales, la administración simultánea de insulina evita al páncreas cualquier esfuerzo indebido, semejante al que observó Allen en sus animales experimentales.

El problema de la composición de la dieta del diabético ha recibido también soluciones diametralmente opuestas. Algunos recomiendan una limitación moderada de los hidrocarbonados y de las proteínas. Otros como Geyelin, Gray y Rabinowitch propugnan dietas de elevado contenido de hidrato de carbono y con pocas grasas, acompañadas de grandes cantidades de insulina, mientras que Joslin y su grupo limitan el ingreso de hidrocarbonados e intentan evitar la glucosuria con cantidades mínimas de insulina. La tendencia durante los últimos cinco años se orienta hacia una dieta normal y algunos llegan hasta permitir una libre elección de alimentos, controlando la glucosuria con insulina. Con los progresos

de la insulinoterapia y el advenimiento de los nuevos tipos de insulina resultan innecesarios los procedimientos complicados que antes se usaban y puede asimismo prescindirse de los llamados "alimentos diabéticos", confeccionadas con sustitutos de glucosa. Hoy en día el tratamiento dietético de la diabetes se ha convertido en un problema de selección y adaptación de la dieta más bien que en un asunto de limitación y restricción.

Una vez determinadas las necesidades calóricas del paciente es necesario la distribución entre las proteínas, hidrocarbonados y grasas deseadas por el médico. Para la distribución calórica utilizamos el siguiente esquema:

1) Hidrocarbonados

   a. 100 - 125 gramos - para el paciente obeso con dieta de reducción sin insulina.

   b. 150 - 200 gramos - Para el paciente en su peso normal que no necesite insulina.

   c. 225 - 300 gramos - para el paciente bajo peso que requiere insulina.

2) Proteínas - 1 a 1.5 gramos por kilo de peso ideal para el adulto.

   Muy pocas veces menos de 90 gramos diarios excepto en dietas de reducción de peso. En el niño diabético usamos de 2 a 3 gramos por kilo de peso ideal.

3) Grasas - completamos el balance calórico, siempre tratando de mantenerlas bajas pero nunca menos de 40 gramos diarios, excepto en las dietas de reducción de peso. Por lo general nunca más de 100 gramos diarios.

En otro tiempo era necesario equilibrar la proporción de grasas a hidratos de carbono a fin de evitar la cetosis. Actualmente esto es de menor importancia, ya que la proporción permitida acostumbra ser baja. Administrando insulina y con la orina aglucosúrica hay realmente poco peligro de acidosis incluso con una proporción de grasas a hidrocarbonados de 3 a 1, si el aporte de proteínas es bajo. Sin embargo, esto no es necesario, ya que fácilmente se preparan dietas casi normales que contienen proteínas, grasas e hidrocarbonados en una proporción de 1:2:2:. (C = 200, P = 90, G = 90).

La dieta del diabético no solo debe ser amplia, sino que debe ser superior en calidad a la de un individuo normal. Además deben administrarse suplementos vitamínicos particularmente los

miembros del complejo B y la Vitamina A en cantidades superiores a las necesarias para el individuo normal, ya que no sólo estas vitaminas son escasas en la dieta restrictiva del diabético, sino que los requerimientos de éste son superiores a los del individuo normal. Asimismo debe prestarse atención a que el suministro de calcio, fósforo y hierro sea adecuado, particularmente durante la edad del crecimiento.

Insulinoterapia

Cuando, a pesar de las restricciones dietéticas, persiste la glucosuria, está indicada la administración de la insulina. En ausencia de complicaciones o de acidosis puede disponerse de algún tiempo para determinar la dosis óptima y el establecimiento del plan terapéutico. En algunos casos es preferible la hospitalización aunque yo personalmente prefiero controlar la mayoría de mis pacientes en status ambulatorio.

Actualmente hay cinco tipos de insulina en el mercado, a saber, regular, cristalina (insulina-zinc), protamina-cinc, globina y NPH. La insulina corriente y la insulina cristalina tienen efectos idénticos ejerciendo su acción máxima hipoglucemiante de dos a tres horas después de la inyección subcutánea. Este tipo de insulina de duración corta y de acción rápida llena su cometido a perfección en el tratamiento de complicaciones cuando se administra cada seis horas durante el día y la noche. Es también de mucho uso como insulina aditiva en conjunción con la protamina-cinc y la NPH.

Tanto la NPH como la globina son insulinas de tipo intermedio, es decir, exhiben sus efectos hipoglucemiantes durante las horas del día y especialmente durante la tarde. La NPH (N - Neutral, P - Protamina, H - Hagedorn) es superior a la globina por diferentes razones, a saber, su estabilidad, la facilidad en que puede mezclarse con insulina regular y su efecto un poco más marcado durante la mañana y la noche. Esto se debe a que la NPH es una mezcla comercial de dos partes de insulina regular y una parte de protamina-cinc sin exceso de protamina en la combinación y a un pH casi neutral. Esta insulina es la preparación indicada en el tratamiento de la diabetes juvenil y en las afecciones severas en el adulto.

Por el contrario, la insulina protamina-cinc actúa lentamente, produciendo un descenso gradual de la glucemia que persiste aún al cabo de veinticuatro o treinta y dos horas después de su administración. Consiguientemente, el peligro de una reacción hipoglucémica tras la administración de esta insulina es mayor durante las horas de la madrugada y antes del desayuno. Esta preparación to-

davía ocupa un sitio de importancia en la terapia del diabético, a pesar de las muchas objeciones levantadas a su uso por varios diabetólogos norteamericanos. Es de valor en algunos pacientes adultos que requieren dosificaciones mínimas de insulina y en otros con tendencia a hiperglucemias nocturnas.

Las necesidades de insulina pueden variar con el tiempo debido a remisiones o exacerbaciones de la enfermedad. El paciente debe analizar su orina frecuentemente a fin de evidenciar una posible glucosuria, especialmente durante cualquier proceso mórbido que pueda complicar su enfermedad. También se le pondrá en guardia contra la aparición de síntomas de un exceso de insulina. Es importante notar que en el diabético bien compensado el ejercicio moderado es ventajoso y puede hacer disminuir rápidamente las necesidades de la insulina.

La cantidad de insulina necesaria para contrarrestar la glucosuria en el diabético depende en gran parte de los hidrocarbonados administrados en la dieta, pero a pesar de que ésta sea perfectamente bien regulada, de vez en cuando se presentan amplias variaciones en los requerimientos de insulina de los pacientes. En algunos casos, cuando la diabetes se ve complicada por otros trastornos, pueden necesitarse enormes cantidades de insulina. Se tiene noticicia de un caso que necesitaba 4,000 unidades diarias de insulina; se trataba de un diabético que padecía de una leucemia linfoidea crónica. Experimentalmente requieren grandes cantidades de insulina los animales hechos diabéticos por la inyección de extractos hipofisarios o de aloxana. Los efectos tóxicos de estas substancias son comparables con la coexistencia de una infección o trastorno que aumente los requerimientos de insulina.

El empleo de la insulina es seguido a veces de atrofia localizada de la grasa subcutánea alrededor del sitio de la inyección. La causa de esta atrofia insulínica es desconocida y ha sido atribuída a la falta de antisepsis o a las repetidas inyecciones en el mismo sitio. Otras veces se presenta una ligera inflamación en el punto de la inyección; esto por lo general es una manifestación alérgica que desaparece espontáneamente al cabo de unos días. Sin embargo, cuando se presenta una reacción más grave y dolorosa es preciso cambiar la marca de insulina; usar insulina cristalina o desensibilizar al paciente. En los individuos caquécticos con diabetes severa, se presenta a veces edema generalizado durante los primeros días de tratamiento.

Pueden presentarse reacciones a la insulina como resultado de errores de dosificación, por irregularidades alimenticias, por dejar de hacer una comida o tras un ejercicio excesivo. Las reacciones producidas por la insulina protamina-cinc acostumbran pre-

sentarse a las horas de la madrugada o antes del desayuno, las producidas por globina o NPH de tres a seis de la tarde y aquellas motivadas por la insulina regular de dos a tres horas después de la inyección. Estas reacciones se deben a la hipoglucemia y están acompañadas por síntomas bien conocidos por ustedes. Debe uno cuidarse de no confundir la inconsciencia de la hipoglucemia con la provocada por el coma diabético y viceversa. El diabético debe tener siempre a mano terrones de azúcar o caramelos que tomará al menor síntoma de hipoglucemia. En el coma hipoglucémico, la inyección endovenosa de veinte a cincuenta gramos de glucosa bastará para que el paciente recobre rápidamente; si ello no es posible, está indicada la administración de jarabe por sonda gástrica. Si existe duda sobre el diagnóstico se debe administrar azúcar por cualquier vía. En el shock insulínico es también recomendable la inyección subcutánea de medio a 1 cc. de epinefrina al 1:1000, siempre que el hígado tenga suficientes reservas de glucógeno o que no sea después de un ejercicio violento.

Para terminar mi presentación quiero decir que quizás en ninguna otra enfermedad es tan esencial como en la diabetes la cooperación del paciente para que el tratamiento tenga éxito completo. Debe enseñarse al paciente la manera de comprobar la presencia de glucosa y cuerpos cetónicos en la orina, como debe regular su dieta y cómo debe reconocer los síntomas de acidosis inminente o de reacción a la insulina. Son recomendables diversas obras de divulgación diabética con instrucciones para el enfermo. También es esencial despertar en el paciente una actitud optimista, así como el sentido de responsabilidad necesario para llevar a cabo el tratamiento de una enfermedad muy fácil de controlar.

STUDIES ON BRUCELLOSIS

A. POMALES-LEBRON, CARLOS FERNANDEZ, M. D. PIZARRO, J. I OTERO and SYLVIA VARGAS.

Department of Microbiology, Division of Bacteriology, University of Puerto Rico School of Medicine - School of Tropical Medicine, San Juan, P. R.

I. THE ISOLATION OF *BRUCELLA* FROM THE BLOOD.

A method described by Ruiz Castañeda in 1947[1] is most commonly used at present for the isolation of brucellae from the blood. Castañeda's double medium has the advantage that reinoculation of the agar surface can be accomplished by tilting the culture bottle bathing the slanted solid medium with the liquid culture, thus eliminating repeated transplants and contamination. This technic has increased considerably the chances of obtaining positive blood cultures in human brucellosis.

In studies concerned with the intracellular localization of **Brucella**, citrated blood was routinely cultured on the surface of solid media before and after desintegration in a Mickle tissue desintegrator. This procedure gave very good results and it has been used successfully in this laboratory during the last three years. Reference to this method was made in a recent publication,[2] but technical details were omitted.

Method. Five ml or more of blood are mixed with an equal volume of 0.9% saline containing 1% sodium citrate. One half ml of citrated blood is spread over the surface of a trypticase or tryptose agar plate.[(a)] The same amount of blood is cultured similarly after 5 min. desintegration under optimal conditions in a Mickle vibrator.[(b)] The blood plasma difuses into the medium leaving phagocytes containing brucellae, or the bacteria themselves, deposited on the surface. This minimizes the inhibitory effect of substances which may be present in the blood. Table I shows the results obtained in a typical experiment using concomitantly the plate method and Castañeda's double medium.

When desintegrated citrated blood was placed directly on the surface of solid media growth appeared earlier than in Castañeda's medium. On agar media, inoculated with equal amounts of desintegrated and undesintegrated blood, growth was more abundant and colonies were usually larger in cultures of desintegrated material. This observation, made with blood obtained from resist-

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(a) Agar plates (medium about 0.3 ml deep) are placed for one hour in the 37°C incubator immediately before inoculation.

(b) Manufactured by H. Mickle, Hamton Middx., England.

TABLE I

Cultures of Undesintegrated and Desintegrated Citrated Blood from Guinea Pigs Infected with *Brucella abortus*.
Numerator: 4 days incubation; denominator: 10 days incubation.

Days after inoculation	No. of colonies					
	Castañeda's method (2 ml.)			Plate (0.5 ml.)		
		D			D	
	U	5 min.	15 min.	U	5 min.	15 min.
57	—	—	?	—	1	—
	—	—	?	—	1	—
57	—	1	—	—	11	10
	M	M	M	—	11	10
57	—	—	—	1	4	1
	—	M	M	1	4	1
57	—	1	?	8	40	29
	M	M	?	9	46	34
57	—	—	—	12	36	60
	M	M	M	12	44	88
57	—	1	?	9	42	23
	M	M	?	9	42	23
186	—	—	?	—	—	—
	—	—	?	—	—	—
186	—	—	?	—	—	—
	—	—	?	—	—	—

U = undesignated, D = desintegrated, M = many, ? = not done.

ant animals as well as from guinea pigs in which agglutinins had not yet developed, suggests that bacteria may have been dislodged from the phagocytes, making the nutrients in the medium more readily accessible. When a vibrator is not available undesintegrated citrated blood can be cultured with good results. Two ml. of citrated blood can be cultured with good results. Two ml. of citrated blood can be cultured if large plates are used.

Cultivation of citrated blood directly on the surface of solid media constitutes an improvement over other methods used routinely in the isolation of brucellae from the blood. The method permits the quantitative estimation of brucellae in the blood. Braun[3]

has recently described a procedure utilizing solid media for blood cultures in brucellosis.

II. RESISTANCE TO REINFECTION IN EXPERIMENTAL BRUCELLOSIS.

This work was intended to determine if a relationship exists between resistance to superimposed infection and the presence of the organisms of primary inoculation in the body of guinea pigs infected with **Br. abortus.** An **anaerobic** strain was used for primary inoculation and an **aerobic** strain for reinoculation. The organisms of reinoculation never persisted in the tissues in the presence of the brucellae of primary infection, except in one instance. The exception was a guinea pig reinoculated 11 days after infection. Some animals, from which the organism of the original infection was not recovered from any of the tissues, were resistant to reinfection. Resistance waned after the organisms of primary inoculation could not longer be recovered from the tissues.

A local reaction with abscess formation was produced at the site of subcutaneous reinoculation in the infected guinea pigs and brucellae were recovered in large numbers from these lesions. The organisms of reinfection penetrated the deep tissues but disappeared promptly. This suggests that the local reaction was not a major contributing factor in resistance to reinfection. When previously infected animals were injected intracardially the brucellae of reinfection also disappeared rapidly from the tissues.

III. SEARCH FOR ANTIBIOTIC SUBSTANCES, ACTIVE AGAINST *BRUCELLA* AND OTHER BACTERIA, IN PUERTO RICAN PLANT MATERIALS.*

Aqueous and alcoholic extracts were prepared from different parts (leaves, roots, flowers, fruit, etc.) of 525 tropical plants growing in Puerto Rico. The activity of crude extracts was tested by the filter paper disc technic in which positive results are shown by areas of inhibition around the discs, saturated with the extract, placed on the surface of agar plates previously inoculated with the bacteria to be tested. The following test organisms were used routinely:

1. Staphylococcus aureus (three strains)
2. Pseudomonas aeruginosa
3. Group A hemolytic streptococcus
4. Streptococcus agalactiae (from bovine mastitis)

* This work was supported by a grant obtained through the Agriculture Experiment Station of the University of Puerto Rico.

5. Proteus sp.
6. Salmonella typhosa
7. Br. abortus
8. Myco. phlei
9. Escherichia coli
10. B. subtilis

Table II shows the results obtained with extracts from 30 different plants.

TABLE II

ANTIBIOTIC ACTIVITY OF PUERTO RICAN PLANT MATERIALS (CRUDE EXTRACTS)

Name of Plant		Test Organisms									
Common	Scientific	M. aureus	Group A Strep.	Strep. agalactiae	S. typhosa	Ps. aerugi-nosa	Proteus sp.	E. coli	Br. abortus	B. subti-lis	Myco. phlei
Acacia	Leucaenea glauca (L) Benth.	12 a/b Aq.	0	0	0	0	0	0	0	0	0
Acalifa	Acalypha Wilkesiana, Muell. Arg.	16 a I, Aq.	0	0	0	0	14 a I, Aq.	0	0	0	0
Aguacate	Persea Persea (L) Cockerell.	0	13 a Alc.	0	0	0	0	0	0	0	8 a Alc.
Almendra	Terminalia catappa L.	0	0	0	0	0	10 a Aq.	0	0	0	0
Astromelia	Lagerstroemia indica L.	10 a/b I, Aq.	0	0	0	0	10 a/b I, Aq.	0	0	0	0
Cardo santo	Argemone mexicana L.	0	7 a/b/g/e Alc.	0	0	0	0	0	0	7 a/b/g/e, Alc.	11 a/b/g/e Alc.
Cohitre blanco	Athyrocarpus persicariafolius (DC.) Bansel.	15 a-b I, Alc.	15 a/b Alc.	12 a/b Alc.	0	0	0	0	0	0	0
Cupey	Clusia rosea Jacq.	20 d Alc.	8 d Alc.	0	0	0	0	0	0	15 d Alc.	25 d Alc.
Espuela de galán	Impatiens Balsamina L.	15 g/d Alc., I	0	0	10 g/d Alc., I	0	0	0	0	0	0
Gandul	Cajan Cajan (L) Millsp.	12 a Alc.	0	0	0	0	0	0	0	0	0
Guayacán	Guaiacum officinalis L.	10 g Aq.	14 g Aq.	14 g Aq.	0	0	0	0	0	12 g Aq.	0
Jaraguasc	Varronia corymbosa (L) Desv.	7 a/c Alc.	0	0	0	0	0	0	0	0	0
Jazmín francés	Moringa Moringa (L) Mills.	25 d Aq.	0	12 d Aq.	0	0			40 d Aq.	0	C
Jobo gusanero	Spondias Mombin L.	15 a/b/g Alc.	0	0	0	0	0	0	0	0	0

Numbers indicate diameter at area of inhibition in mm. a—leaves, b—stem, c—bark, d—seed, e—fruit, f—root, g—flowers, f—acorn, Alc.—Alcoholic extract, Aq.—aqueous extract, I—incomplete inhibition, O—negative.

TABLE II (Cont.)

ANTIBIOTIC ACTIVITY OF PUERTO RICAN PLANT MATERIALS (CRUDE EXTRACTS)

Maga	Montezuma speciosissima Sessé & Moç.	21 e Aq.	10 e Aq.	10 e Aq.	0	0	0	0	12 e Aq.	12 e Aq.	10 e Aq.
Malagueta	Amomis coryophyllata (Jacq. Krug & Urban.)	0	0	0	10 a/c I, Alc.	0	10 a/c Alc.	0	10 a/c I, Alc.	0	0
Malva	Malachra capitata L.	20 a/ b Alc.	15 a/b Alc	0	0	0	0	0	0	0	0
Mamey	Mammea americana L.	20 a Alc.	10 a Alc	0	0	0	0	0	50 a Alc	0	12 a Alc.
María	Callophyllum antillanum Britton	0	10 a/b Aq.	0	0	0	0	0	0	0	0
Panapén	Artocarpus communis Frost	10 a Aq.	0	0	0	0	0	0	0	0	0
Papaya	Carica papaya L.	0	0	0	0	0	0	0	0	0	8 a Alc.
Pomarrosa	Jambos Jambos (L.) Millsp.	15 a/b Aq.	0	0	0	0	15 a Aq.	0	0	0	14 a Aq.
Pomarrosa malaya	Jambos malaccensis (L.) DC.	10 a/c Aq.	0	0	0	0	10 a/c I, Aq.	0	0	0	10 a/ c I, Aq.
Resedá blanca	Roseda odorata L.	10 a/g Alc.	0	0	0	0	0	0	18 f Alc.	0	0
Tamarindo forastero	Vangueria madagascariensis Gmelin	0	0	0	0	0	0	0	0	0	8 a Alc.
Verbena	Valerianoides jamaicense	15 a/b Alc.	15 a/b Alc.	10 a/b Alc.	0	0	0	0	0	0	0
Verdolaga	Portulaca oleracea L.	15 a/b Alc.	10 a/b Alc.	0	0	0	0	0	0	0	0
Yautía de jardín	Cyrtospadix bicolor (Ait.) Britton and Wilson	0	0	0	0	0	0	0	0	0	7 a/ b Aq.
"Bottle brush"	Callistemon lanceolatus	25 a Alc.	0	0	0	0	0	0	0	12 a Alc.	30 a Alc.
Bijao	Alpina exaltata (L.) R. & S.	8 a/b/h Alc.	0	0	0	0	0	0	0	0	0

Numbers indicate diameter at area of inhibition in mm. a—leaves, b—stem, c—bark, d—seed, e—fruit, f—root, g—flowers, f—acorn, Alc.—Alcoholic extract, Aq.—aqueous extract, I—incomplete inhibition, O—negative.

Alcoholic extracts of the seed of **Moringa Moringa** and the leaves of **Mammea americana** are prominently active against brucellae.

Table III shows the effect of extracts of **Mammea americana** on different strains of **Brucella, Pseudomonas aeruginosa** and group B streptococci of human and bovine origin.

TABLE III

Effect in vitro of crude alcoholic extract of *Mammea americana* on *Brucella*, *Pseudomonas aeruginosa* from human urinary infections and Lancefield's group B streptococci of human and bovine origin.

Organism	Number of strains Tested	Susceptible	Resistant
Br. abortus	10	10	0
Br. melitensis	12	12	0
Br. suis	6	6	0
Group B streptococci			
Bovine	73	30	43
Human	21	18	3
Pseudomonas aeruginosa	66	2	64

All **Brucella** strains were susceptible to the antibiotic substance obtaining in the leaves of **Mammea americana.** Pseudomonas aeruginosa was usually resistant and 59% of bovine strains of group B streptococci were resistant as compared to 14% of resistant strains among those from human sources. It must be noted that the test culture of **Streptococcus agalactiae** (group B) used routinely was resistant to the active principle in **Mammea americana** (See Table II). This illustrates the limitations of work of this nature when only one test strain of a particular bacterial species is used.

Extracts made with ethanol, methanol, pyridine, ethyl ether, chloroform, ethyl acetate and acetone were equally active. Aqueous extracts were inactive. Powdered dry leaves kept at room temperature for two years yielded alcoholic extracts of apparently undiminished potency. The alcoholic extracts of **Mammea americana** is also active against **M. aureus, B. subtilis** and **Myco. phlei.**

The crude greenish alcoholic extract of **Mammea americana** is toxic for mice. When the crude preparation is absorbed with norite A and filtered through paper, a water clear active filtrate is obtained which is not toxic for mice. Preliminary experiments suggest that the alcoholic extract after charcoal absorption confers some protection to mice inoculated intraperitoneally with an 80% lethal dose of **Brucella abortus.** This stable active principle obtaining in the leaves of **Mammea americana** is worthy of further study.

The crude alcoholic extract obtained from the seed of Moringa Moringa is toxic for mice when given intraperitoneally, however active extracts prepared from the flower are not toxic. This suggests that the antibiotic and toxic substances may be different.

The alcoholic extracts of the seed of **Clusia rosea** and the leaves of **Callistemon lanceolatus** are prominently active against **Myco. phlei.** The action of these preparations on other acid fast organisms, including tubercle bacilli, must be investigated further.

In this preliminary report only the most important results of this survey have been recorded; description of technics and other details have been omitted. A detailed account of this work will be published elsewhere.

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SEVERE NUTRITIONAL ANEMIA IN PREGNANCY

MANUEL FERNANDEZ FUSTER, M.D.,*
CELSO RAMON GARCIA, M.D. and
A. A. CINTRON-RIVERA, M.D.*

In this clinic, and in Puerto Rico in general, severe nutritional anemia associated with pregnancy is seen with moderate frequency. These patients present such a striking clinical syndrome that a study of this condition was undertaken by one of the authors (M.F.F.) during the past several years and is being continued at the present time.

This syndrome, which is more often encountered in the early puerperium, presents a picture of a severely ill patient with marked anemia, fever, leucopenia and diarrhea. Shortly after embarking on this study, it soon became apparent that the anemia was not due to actual blood loss during labor but rather that it had developed during the pregnancy. These anemias are initiated or aggravated by the pregnancy with the increased demands of the fetus upon the maternal reserves.

This clinical entity is often referred to as the pernicious anemia of pregnancy and only sporadic references appeared in the literature during the first quarter of this Century. The first large series presented were those of Balfour[1] and McSwiney[2] in 1927. They reported independent series of 150 and 43 cases, respectively which they collected over a period of 2 and 2-1|2 years, respectively. In the continental United States, this condition is not very common and the low incidence undoubtedly is explained by the higher level of nutrition together with the more adequate facilities for medical care. For this reason, the American literature contains relatively few reports on this subject and these usually are comprised of reports of single cases or of but a few cases.

CLINICAL FEATURES

The cases encountered in this study presented the following clinical features:

1—**Race** - The patients in this series were more often white than colored. This ratio was the reverse of the ratio in general census of the clinic. According to the literature,[1,2,3] the Hindu, also appear to be susceptible.

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* From the Department of Obstetrics and Gynecology and the Clinical Research Laboratory of the School of Medicine, University of Puerto Rico and the San Juan City Hospital, Santurce, P. R.

2—**Economic Status:** The clinical picture is associated with poverty, with overcrowding, poor diet, ignorance and inadequate medical care. It is never seen in private practice.

3—**Diet and Nutrition** - It is our firm conviction that the disease is essentially due to a nutritional failure. The poor diet and nutrition can generally be traced to either one or more causes, such as sheer poverty with inability to maintain an adequately balanced diet, food fads, and vomiting of pregnancy. Vomiting of pregnancy may initiate a vicious cycle because the patient will limit the diet because of the anorexia. This will debilitate the patient further and vertigo will ensue which in turn will promote further nausea and vomiting, etc. Anorexia nervosa is also encountered as a causative factor, on rare occasions. This syndrome appears even after a short period of inadequate nutrition. The inadequate nutrition superimposed upon the increased nutritional requirements during pregnancy rapidly deplete the maternal reserve. A mixed type of anemia is usually noted as evidenced by the frequent association of a megaloblastic picture with hypochromia. Hemoglobins as low as 1.6 gms. (Sahli) have been noted.

The following is a typical case history:

The patient is a poor pregnant woman who started with vomiting early in pregnancy. Because of the vomiting of pregnancy the patient limited the food intake to a liquid concoction of poor nutritional value. As the patient became weaker she complained of vertigo with subsequent nausea and vomiting. The patient then developed stomatitis, diarrhea, edema and eventually became prostrated. On occasion the extreme cachexia of malnutrition is often masked by the generalized edema.

4—**Diarrhea and Vomiting** - As pointed out above, vomiting, which is frequently encountered in this disease, plays an important role in the etiology of the nutritional symptom. In many cases, however, diarrhea, appearing during the antepartum period or shortly after delivery, may be the prominent symptom. When appearing in the puerperium it is usually very severe. This diarrhea, usually watery and greenish in color, is striking because of the frequency and force of the bowel movements.

5—**Pyrexia** - The development of a fever is a very common and striking sign of this syndrome which may develop during pregnancy, but more likely in the early puerperium, usually during the 1st 24 to 48 hours. It varies in severity

and is usually of the continuous type. Usually, the temperature returns to normal, as if by lysis, under proper treatment, but on occasion it may be persistent for several months. A "step ladder" drop is often encountered with each successive blood transfusion administered. The rapid disappearance of the fever, upon treatment of the patient with Folic Acid, has been pointed out by Birks.[4]

The etiology of the fever is obscure. Although the temperature rise might be classified as infectious in origin, this is probably not the case. This fever is of a more continuous type than that encountered in septic conditions and has its onset earlier than one would expect in the case of puerperal sepsis. Furthermore the signs and physical findings of pelvic or other infection were absent and there was little response to the administration of antibiotics and other chemotherapeutic agents. Some animal experimentation[5] may shed light on this perplexing problem. The normally fatty marrow of the tail bones of the rat becomes hemopoietic when its temperature is raised such as by surgically inserting part of it into the peritoneal cavity. The cooler section remaining outside the body is unchanged. It may be possible that the pyrexia of these patients may be a defense reaction in an attempt to increase hematopoiesis. The fever usually disappears within a few days after starting the anti-anemic therapy, alone.

6—**Course** - The cases, untreated during pregnancy, tend to become progressively worse. These patients have rapid, premature labors with slight blood loss. Nevertheless, in the more severe cases, acute heart failure, with death, may occur a few hours after delivery. Even in the mild case that survives the first 24 hours, the more severe syndrome of fever, diarrhea, glossitis and megaloblastic anemia with leucopenia is likely to develop. The prognosis without specific treatment is grave but recovery after prolonged illness has been seen. With proper therapy, however, recovery is rapid and it is striking how quickly one of these severely cachectic individuals recovers. The newborns, however, are frequently stillborns. Their prematurity and generalized weakness are contributory to the high neonatal death rate. The development of megaloblastic anemia in these infants is noted.[6,7,8]

7—**Prognosis:** Most deaths occur incident to heart failure. This is frequently seen in those severely anemic cases which go into labor and deliver before improvement can be realized. These patients may expire, in heart failure, a few hours after delivery. To obviate this, early transfusion of the

severe cases is desirable. However, caution must be exercised as these patients tolerate transfusions of the order of 500 cc (or more) rather poorly, since the increased blood volume may be too great a strain on the apparently weakened myocardium. This may result in acute heart failure and death. The transfusion, moreover may give rise to an undesirable situation since it may be responsible for a transfusion reaction, even though minor, which is usually followed by the premature onset of labor. The slow administration of 200-300 cc of whole blood (and preferably diluted in isotonic solution) is recommended in an attempt to avoid these sequelae. Early treatment with the specific hematinics obviate the possible dangers of the transfusions. On occasions, progression to aplastic anemia may occur.

LABORATORY FINDINGS

1—**Red Blood Cells:** The severe cases usually exhibit red blood cell counts of less than 2.5 million per cu. mm. with a hemoglobin of 6.5 gms. or less. The cells are usually macrocytic with a Mean Corpuscular Volume of 100 or more, although on occasion these may be normocytic or, in even rarer instances, microcytic. Moreover, inasmuch as this condition is associated with multiple nutritional deficiencies, hypochromia, normochromia as well as hyperchromia may be seen. By far, the most common combination appears to be a macrocytic hypochromic anemia. The reticulocytes are either absent, normal, or slightly above normal. The varied and mixed hematological findings has been noted previously by other investigators (Davidson and Davis).[9]

2—**Leucocytes:** The cases usually show a leucopenia which varies with the severity of the anemia and there is a tendency toward agranulocytosis.

3—**Bone Marrow:** The bone marrow picture varies with the severity of the disease and shows a megaloblastic response.

4—**Gastric Acidity:** All patients on whom gastric analysis were performed, show the presence of free HCL, either prior to or after Histamine stimulation.

5—**Plasma Proteins:** There is a decrease in the total proteins particularly of albumin. This hypoproteinemia is more marked in the edematous patients.[3]

TREATMENT

In 1934, Wills[10] reported on the efficacy of autolyzed yeast in the treatment of these severe anemias of pregnancy. We concur with those who have noted the lack of response with the use

of Liver extract in these cases[4,11,12,13,14,15,16]. Wolff and Limarzi[17] reported satisfactory hemapoietic response with liver extract and transfusions. We are inclined to feel that most of the effect was obtained thru the use of the transfusion rather than the liver extract. There are reports of the ineffectiveness of response thru the use of transfusions in this condition,[12,16] however it has been our experience that the use of small, repeated transfusions must be used (at least 2 to 3 per week) to obtain the desired results. Folic Acid appears to give a very dramatic improvement in this condition and appears to be the treatment of choice. This has also been substantiated by others as reported in the literature.[4,7,11,12,13,14,15,16,18,19,20] Response appears satisfactory in doses of 5-15 mg daily although Allen[12] reported having to use very large doses (75-135 mg) to obtain a satisfactory response in severe cases in India. The response to folic acid is rapid and the reticulocyte rise reaches a peak in 5-7 days in most instances. This is accompanied by the remission of the pyrexia and diarrhea together with marked improvement in the general well being of the patient. A very favorable sign is the development of a voracious appetite but a few days after the initiation of this therapy. In those cases showing iron deficiency, ferrous sulfate is administered also.

The place of Vitamin B_{12} in the treatment of this condition appears to remain unsettled. Several investigators[3,21,22,23] have reported conflicting opinions concerning the efficacy of Vitamin B_{12} in the treatment of these macrocytic anemias. Goldsmith has indicated that orally administered Vitamin B_{12} is not as effective as by the parenteral route. Our present studies appear to agree with these findings. The oral administration of 15 mcg of Vitamin B_{12}, daily, does not appear to produce a change in the reticulocyte count but the same intramuscular administration does appear to cause a response in some of the cases. In one of our cases, Beclysyl 5% in water (Abbott) was administered for fluid replacement in connection with the diarrhea. This was followed by a prompt and dramatic remission of symptoms and a marked rise in the reticulocyte count. Beclysyl (Abbott) is comprised of: Dextrose 50 gm., Na cl 9 gm., thiamine Hcl 10 mg., Riboflavin 10 mg., Nicotinamide 250 mg., Pyrodoxine Hcl 5 mg., B_{12} 3 mcg. 1000 cc fluid.

It is quite possible that the intravenous administration of small quantities of Vitamin B_{12} may give a more marked and more dramatic response. This warrants further study.

CONCLUSIONS AND SUMMARY

1—The clinical syndrome of severe nutritional anemias associated with pregnancy has been reviewed.

2—The therapy for this condition is still unsettled, however,

the administration of an adequate well balanced diet together with the use of repeated small transfusions or the early use of Folic Acid appears to be most effective.

3—Inasmuch as this is a preliminary study, no conclusions are drawn concerning the effectiveness of Vitamin B_{12} in the treatment of this condition. Further investigation is being undertaken to elucidate this point.

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FOLIC ACID AND ENDOGENOUS BACTERIAL INFECTION

(STUDIES IN THE RAT)

CONRADO F. ASENJO*

The most dramatic finding in the blood of folic-acid-depleted rats is a severe leucopenia and granulocytopenia. In this respect the rat differs from the human as in this latter species folic acid deficiency results in a severe macrocytic hyperchromic anemia.

In 1944, Gross, Axelrod and Bosse reported for the first time gross lesions in the liver and spleen of rats fed a diet low in folic acid and containing between 0.5 and 1% sulfaguanidine as inhibitor. These lesions were prevented, and in many instances cured, by the oral administration of whole liver or of a folic acid concentrate plus biotin. At the time they performed their work (1944), folic acid had not yet been isolated in a pure form.

In 1947, we (Asenjo '48) observed gross lesions in the spleen of folic-acid-depleted rats similar to those described by Gross, et al ('44). To a much lesser extent, we also observed lesions in the liver although they were not reported at the time. The synthetic ration used by us was folic-acid-free and contained as an inhibitor either 2% succinylsulfathiazole (SST) or 0.85% x-methyl folic acid. The incidence of splenic lesions in the depleted animals was of over 80% in either case. The animals on the SST containing diets responded to treatment with folic acid, and the lesions were either prevented or cured; however, in the case of the x-methyl folic acid diet, it was impossible to overcome the inhibition when folic acid at a level effective in the case of the SST diet was fed. This is in agreement with the findings of Franklin, et al, ('47) that the inhibition ratio of x-methyl folic acid in the rat is of the order of 3000:1.

Later on, Philips and Thiersch ('49) and Kodicek and Carpenter ('50) reported the presence of similar lesions in rats receiving as inhibitors aminopterin and SST, respectively. It is interesting to note that these investigators, as well as Gross, et al ('44) obtained a high incidence of spleen and liver lesions principally in animals with chronic folic acid deficiency, while the initial high incidence of spleen lesion observed by us was in animals that had developed an acute deficiency in a period of about 31 days.

Nature of the lesion. In the rest of this discussion we will refer exclusively to the splenic lesions, which are the gross pathological manifestations encountered most frequently by us in the

* Department of Biochemistry and Nutrition, School of Medicine, School of Tropical Medicine, University of Puerto Rico, San Juan.

folic-acid-depleted rats. Histological examination of the abnormal spleens revealed necrotic portions sharply demarcated from the more normal, but intensely congested parts. In the necrotic areas there were large spreading colonies of bacilli, which were scattered not only in the pulp, but also within the venous thrombi. It is reasonable to conclude that these lesions are the end result of a bacillary infection which damaged the venous walls facilitating the formation of a thrombus followed by infarction. Direct smears made from material dissected out of the necrotic areas showed massive amounts of bacteria. Blood agar and thioglycollate broth cultures from the infarcted spleens developed abundant growth of irregularly staining pleomorphic diphtheroid forms, gram positive or gram negative cocci or gram negative coli-like rods (Asenjo, Quintana and Pomales-Lebrón '52).

These findings were all in agreement with those of Gross, et al ('44), except that we observed, in all cases studied bacteriologically, i.e. animals killed as well as those that died spontaneously, the presence of bacteria in the splenic lesions, while they reported the finding of bacteria only in those animals that had died spontaneously. This, in addition to the fact that there was a total absence of tissue reaction to the presence of bacteria, made Gross, et al ('44) conclude that the bacterial invasion must be considered an agonal or post mortem phenomenon. On the other hand, we found bacteria in the infarcted spleens of very active folic-acid-deficient animals. The spleens were removed under aseptic conditions from etherized animals and cultures made (Asenjo, Quintana and Pomales-Lebrón '52). Regarding the lack of inflammatory reaction to the presence of bacteria and the development of necrotic tissues, we believe these to be due to the leucopenia and granulocytopenia predominant in the folic-acid-depleted rats, since little if any reaction could be expected from animals having only a few granulocytes in their blood.

Polymorphonuclears were totally absent in histological sections of spleens from animals kept after depletion of folic acid for periods ranging between 1 to 5 weeks without any supplementation of folic acid or receiving minimal dosages of 0.25 ug and 0.5 ug per day. On the other hand, in those animals that were supplemented with 0.75 ug or more of folic acid per day, polymorphonuclears were found in the spleen sections examined. The incidence of gross infarcts, as well as microscopic necrosis and thrombi, was greatly reduced when 0.75 ug per day of folic acid was administered as supplement. No lesions, gross or microscopic, were observed in the spleen of depleted animals that had received 1.25 ug or more of folic acid per day as supplement for periods of at least five weeks (see table 1).

TABLE 1

HISTOLOGICAL EXAMINATION OF THE SPLEEN OF FOLIC ACID DEPLETED RATS SUPPLEMENTED WITH DIFFERENT LEVELS OF FOLIC ACID

Folic acid supplement* ug. per day	Number of spleens examined	Average number of days on basal diet when animals died or were sacrificed**	Microscopic lesions observed in a single section of the spleen Number of spleen showing:				% of rats showing polymorpho-nuclears in the spleen
			Necrosis only	Thrombi only	Necrosis and Thrombi	% with lesions	
none	30	33 (24-26)***	2	2	11	50	0
0.25	7	89 (35-175)	2	0	0	29	0
0.50	15	96 (38-198)	2	0	2	27	0
0.75	7	125 (57-191)	1	1	0	29	100
1.25	13	86 (63-70	0	0	0	0	100
5.00	14	94 (63-138)	0	0	0	0	100
20.00	6	110 (91-127)	0	0	0	0	100
50.00	5	127 (91-139)	0	0	0	0	100

* The animals started to receive the supplements after having been on the depletion diet for 4 weeks.

** All the rats started to receive the basal folic acid free diet + 2% SST when 21 days old.

*** Minimum and maximum number of days.

In no other nutritional deficiency, as far as we know, have splenic lesions of this type been found. The lesion was prevented or healed in a substantial number of the animals suffering from this deficiency after the administration of folic acid. This was established by laparotomies (Gross, et al '44) and by indirect evidence (Asenjo '48).

Philips and Thiersch ('49) reported that 5% of rats receiving 4-amino PGA (aminopterin) by chronic administration developed salmonella infection. They concluded, after gut lesions were established, that an ascending infection took place with enlargement of the mesenteric lymph nodes, abscess formation, multiple fibrinoid necrosis in liver, spleen, kidney and lung, bronchitis and bronchopneumonia. The gut lesions consisted of extensive desquamation of mucosa, intestinal edema, infiltration of leucocytes, and in some instances, small areas of hemorrhages due to loss of the superficial parts of the plicae. Aminopterin is well known to produce, after repeated administration, severe lesions in the gastrointestinal canal, both of man and animals.

In 45% of our folic acid depleted animals tongue lesions were found (Franklin, et al ('47) and an almost general finding was a yellowish distended intestine.

It is interesting to note that neither the administration of 1 ug. of vitamin B_{12} per day (Asenjo '50) nor of 3 mgs. of aureomycin per day orally (Asenjo and Pomales-Lebrón '52) prevented the development of the splenic lesions.

Observations made in the spleen of 270 depleted folic-acid rats. If we arrange in chronological order the negative control folic acid depleted rats in groups of 30 animals each, the incidence of gross splenic infarct-like lesions observed decreases more or less consistently, from a maximum of 82% in the first group to about 10% in the last group (see table 2).

The reason for this reduction in the incidence of splenic lesions in the latter groups of folic-acid-depleted rats is unknown to us. A marked reduction in the incidence of infarct-like lesions was observed for the first time on July 1949 and this trend has continued until the present time. These animals exhibit all other characteristic signs of folic acid deficiency such as leucopenia, granulocytopenia, cessation of growth, etc., in the usual period of time of about 4 weeks. This seems to indicate that a folic acid deficiency alone is not the only cause of splenic lesions. As the strain of animals used (Wistar S.T.M.) as well as the environment and handling have been the same since we started to work on this problem 7 years ago, one has to suspect the diet as a possible responsible agent.

TABLE 2

INCIDENCE OF GROSS INFARCT-LIKE LESIONS IN THE SPLEEN OF FOLIC-ACID-DEPLETED RATS

Group No.	Dates	Number of rats in each group	Average incidence of gross infarct-like-splenic lesions %
1	Feb. '47 - Feb. '48	30	82
2	Feb. '48 - May '48	30	77
3	May '48 - Aug. '48	30	57
4	Aug. '48 - Dec. '48	30	74
5	Dec. '48 - Dec. '49	30	40
6	Dec. '49 - Mar. '51	30	17
7	Mar. '51 - Dec. '51	30	10
8	Dec. '51 - Aug. '52	30	10
9	Aug. '52 - Jan. '53	30	10

In table 3 is presented the composition of the folic acid free basal diet. This diet, as can be seen, is composed largely of chemically pure substances except for the corn oil, hydrogenated oil and vitamin free casein. Of these three ingredients in the diet casein was probably the least uniform in composition and purity. Therefore, the effect of subjecting this particular ingredient to further purification on the incidence of spleen lesions was investigated.

Vitamin-free casein. The infarct-producing action of a recent batch of vitamin-free casein was compared with that of the same casein but which had been previously submitted to extraction in a Soxhlet for 8 hours with 95% alcohol followed by washing with one liter of distilled water by extraction for 8 hours with 50% alcohol and for another 8 hours with 95% alcohol. The casein thus purified was air dried and used in the preparation of a diet having a composition similar to that indicated in table 3. Two groups of rats were used. In the one receiving the diet containing the treated vitamin-free casein there were 6 animals. In the other group receiving the untreated vitamin-free casein there were 15 rats. All the animals from both groups developed the characteristic signs of folic acid deficiency in the usual period of 4 weeks. However, none of the 15 animals receiving the untreated purified casein developed

TABLE 3

COMPOSITION OF THE BASAL FOLIC ACID FREE DIET

Sucrose	599 gm.
Vitamin-free casein	180 "
Salt mixture	40 "
Cellu flour	40 "
Hydrogenated vegetable oil	100 "
Corn oil (containing 52,000 I.U. vit. A, 13,000 I.U. vit. D, and 32 mg a-tocopherol	20 "
Succinylsulfathiazole	20 "
Choline chloride	1 "
Inositol	8 mg.
Nicotinic acid	40 "
Calcium pantothenate	44 "
Thiamine Hydrochloride	8 "
Riboflavin	16 "
Pyridoxine hydrochloride	8 "
p-aminobenzoic acid	4 "

gross lesion in the spleen. On the other hand, 4 out of the 6 receiving the treated purified casein developed severe lesions in the spleen.

This preliminary experiment encouraged us to repeat the test using two groups of 40 rats each, with the object of obtaining statistically significant data that would permit us to prove or disprove the working hypothesis that some batches of casein contained an unrecognized factor that protected folic-acid-depleted rats from developing infarct-like lesions in the spleen.

Under the conditions of the experiment carried out with a statistically significant number of rats, only 4 rats in the group receiving the treated vitamin-free casein and 5 in the group receiving the untreated vitamin-free casein developed infarcts in the spleen, that is, 10 and 12%, respectively. These results tend to indicate that those obtained in the exploratory experiment were due to chance, and that the treatment to which the casein was submitted does not affect the incidence of spleen lesions.

Blood studies. Asenjo and Pomales-Lebrón ('53) conducted bacteriological studies in the blood of a large number of folic-acid-depleted rats receiving 2% SST as inhibitor in the basal diet. The rats were etherized and blood was obtained by heart puncture under aseptic conditions. In the case of those animals in which cultures of spleen and livers were also performed, they were sacrificed after the blood sample was taken and the organs removed

under aseptic conditions. Microorganisms were recovered from the blood of 18% of the folic-acid-depleted animals. Thirty-seven per cent of the spleens and 24% of the livers examined also contained bacteria. No positive cultures were obtained from rats similarly treated but receiving 50 ug. of folic acid daily. **Non-hemolytic streptococci, Escherichia coli** and **Pseudomonas aeruginosa** composed nearly 75% of the organims isolated from the blood and viscera of the folic-acid-depleted rats. As a characteristic finding in folic-acid-depleted rats is a distended intestine and lesions in the tongue, there is a high probability that the mouth and intestine served as the main portal of entry for the invading microorganisms.

CONCLUSIONS

Considering that folic acid is required for the normal formation of antibodies in the rat, as has been demonstrated recently by Axelrod and his group ('51), and that in addition the blood of folic-acid-depleted rats is almost devoid of phagocytes, it should not be surprising, as indicated by the investigations reviewed in this paper, to frequently find endogenous bacterial infections in folic-acid-depleted rats.

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The author is indebted to Dr. Enrique Koppisch, Head of the Department of Pathology for his continuous interest in this work and for examining a large number of sections. To Dr. Américo Pomales-Lebrón for his advice and collaboration in some phases of this investigation. To Dr. Eleanor Roverud for her effective help in tabulating the microscopic observations. To Dr. Alwin M. Pappenheimer (Harvard University) for examining microscopically a few severely infarcted spleens. Last but not least to my two assistants Mrs. M. L. Martínez and Miss D. Fernández for their effective cooperation.

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PRESENT STATUS OF INTESTINAL PARASITISM IN CERTAIN AREAS OF PUERTO RICO

JOSE F. MALDONADO and JOSE OLIVER-GONZALEZ*

In spite of the strides taken towards the betterment of conditions in this island, the problem of parasitism still remains of great concern. It appears as if in this aspect we have improved very little from conditions existing a decade or more ago (Weller and Dammin, 1945; Hoffman and Faust, 1934).

The accompanying table illustrates the extent to which various intestinal parasitoses occur among 6150 individuals examined during the last year, a great majority of them belonging to the lower economic levels. This work was undertaken under the auspices of the U. S. Department of Defense.

The figures must be taken conservatively, since they are based only upon 2 direct saline smears plus a concentration by the Sodium Sulphate-Triton-Ether centrifugation method, which in general uncover part of the actual cases (Maldonado, Acosta-Matienzo and Vélez-Herrera, 1954). Thus the true situation must still be more accentuated.

	% Children (less than 15 years)	% Adults (over 16 years)
Schistosoma mansoni	23.7	19.5
Trichuris trichiura	92.0	75.5
Hookworm	27.2	24.5
Ascaris lumbricoides	31.0	14.1
Strongyloides stercoralis	7.0	9.0
Endamoeba histolytica (cysts)	15.4	16.1
Endamoeba coli (cysts)	37.1	32.8
Endolimax nana (cysts)	26.1	26.2
Iodamoeba butschlii (cysts)	4.9	5.1
Giardia lamblia (cysts)	17.1	4.9

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* Microbiology Department, School of Medicine, San Juan, Puerto Rico.

STAGE ONE IN THE DEVELOPMENT OF THE SCHOOL OF MEDICINE OF THE UNIVERSITY OF PUERTO RICO

E. HAROLD HINMAN, M.D.*

With the graduation of the first class from our School of Medicine on June 1, 1954, stage one in the development of our School is completed. The writer (1953) traced, in this journal, the origins of this institution including the foundation of the University on March 12, 1903, whose original charter included provision for the establishment of a "Department" of Medicine, the creation of the Anemia Commission of Puerto Rico, the Institute of Tropical Medicine, and Hygiene 1912, the School of Tropical Medicine 1923 and the transition of this in 1949 to a four year School of Medicine.

In this remarkable evolution it is difficult to assay the contributions of single individuals or groups of individuals. It is dangerous to recognize one without the certainty of overlooking others that may be equally important. Since the objective has been to create an American type School of Medicine, it is natural that some bias would enter into my personal effort and also my recent arrival on the scene disqualifies me as an impartial appraiser. Despite these handicaps, I venture to single out a few individuals and groups as having made major contributions towards the development of the proper academic, scientific, and professional environment within which a successful modern medical school might evolve.

Previously (Hinman, 1953) I have indicated the significance of Ashford's contribution. His dedication to a scientific career, his adoption of Puerto Rico as a permanent home and site for his investigations and his acceptance by the Medical profession and the public of Puerto Rico, his ability to strengthen the ties between American Medicine and Puerto Rican Medicine while at the same time recognizing that medicine as practiced in the Continental United States was inadequate for the solution of many health problems peculiar to Puerto Rico; his administrative vision in recommending the establishment of the Anemia Commission, followed by the Institute of Tropical Medicine and subsequently by the School of Tropical Medicine are indicative of his genius, quite apart from the merits of his scientific achievements. In the evolution of the School of Tropical Medicine the splendid cooperation of Columbia University College of Physicians and Surgeons was truly significant.

The overall progress of the University of Puerto Rico in the

* Dean, School of Medicine, University of Puerto Rico, San Juan, P. R.

period 1903 to the initiation of the School of Medicine provided a strong foundation upon which to develop the new school and an academic climate favorable for its growth. For many years graduates of the University had successfully competed in the best medical schools of the Continental United States and the University had long been on the list of Colleges and Universities approved by the American Medical Association for premedical training. The progressive administration of the University, including the Superior Educational Council, and its willingness to make the necessary financial sacrifices to establish a first-rate medical school and their success in convincing appropriating bodies merit recognition for their wise planning. Once the decision to establish the Medical School was made the vigorous action of the University's Chancellor made possible its opening within the short space of one year. The appointment of Dr. Harold W. Brown as Special Assistant to the Chancellor, in charge of Medical Affairs, was an important factor in expediting the establishment of the School and in getting the program under way. His tireless energy and devotion to the project will be recognized in the first graduation exercises involving medical graduates through the conferring of the degree of Doctor of Laws, Honoris Causae.

Our School of Medicine has been most fortunate in the recruitment of its faculty (See appendix). The existing staff within the School of Tropical Medicine has provided a nucleus for most of our preclinical departments as well as for the major clinical fields. Very substantial expansion has occurred and whole new departments have been assembled. While it is our desire to always maintain a hard core of full-time staff in all major clinical departments many of the specialties include only ad honorem appointments. Not only have our staff assumed a heavy teaching load but have undertaken an expanding program in the research field with special emphasis on problems pertaining to tropical and preventive medicine for which Puerto Rico provides unique opportunities. The medical profession of Puerto Rico, by their devoted service to the School of Medicine has been of inestimable assistance. Their continued cooperation is solicited. American Medicine has become a tradition on this island and our school looks forward to a full share in the perpetuation of the highest type of professional activities.

I am particularly proud of the contribution which our student body has made in the evolution of our institution. I especially salute the first graduating class (See Plates I and II). It is a fine group—the equal of other classes graduating from long established schools of medicine but it is unique in several respects. In the first place they are nearly all Puerto Ricans, who received all of their edu-

PLATE I

First graduating class, School of Medicine of Puerto Rico.

PLATE II

First graduating class, School of Medicine of Puerto Rico.

cation in Puerto Rico (with few exceptions) and their training from a predominantly Puerto Rican faculty. They are bilingual having received their formal medical education in English but having all communication with patients in Spanish. I have no reservations concerning their ability upon entering Medical School as compared to that of other Medical School freshmen. They competed, on an equal basis, in the Medical College Admission Test. As medical students many of them have taken the National Medical Board Examinations and despite unfavorable timing of the examinations, have acquitted themselves ably. A few have taken a single clinical clerkship at a sister institution (Columbia College of Physicians and Surgeons) and have done outstanding work. The ready acceptance of one half of our graduating class as interns in leading hospitals in Continental United States further attests to the ability of our graduates.

Another unique characteristic of our student body is that 131 out of 183 have government scholarships. The obligations incurred by the holder of a scholarship for service in a government hospital, health center or in a small community should promptly provide the Department of Health with a reservoir of well trained candidates. The place of women in Puerto Rican medicine has not yet clearly evolved. Our School of Medicine has 33 women among its 183 students a percentage of 18.0 as compared to a national average of less than 6 per cent. We expect our women graduates to justify this unusually high selection ratio.

You are all familiar with our facilities on Ponce de León Avenue adjoining the Insular Capitol (See Plate III). The adaptation has not been difficult of the building of the School of Tropical Medicine as headquarters for the Administration of the School of Medicine and the six departments housed therein (Anatomy, Biochemistry and Nutrition, Physiology and Pharmacology, Microbiology, Pathology, and Preventive Medicine and Public Health), classrooms and laboratories, also dormitory and cafeteria facilities for 151 medical students, library, animal quarters and certain special facilities such as the Blood Bank, Puerta de Tierra Health Unit, etc. The principal teaching hospital is the San Juan City Hospital (see Plate IV) and this institution has cooperated wholeheartedly with us. From their limited space they have provided headquarters for each of the major clinical departments as well as conference rooms and student laboratories. On the grounds of this hospital, a small teaching outpatient dispensary building, with student and research laboratories, 24 examining rooms and a major air conditioned amphitheater has been constructed by the University. It is a genuine pleasure to acknowledge the cooperation of the Government of the Municipality of San Juan and their very substantial

PLATE III

School of Medicine of the University of Puerto Rico.

contribution to the clinical teaching program for our medical students. Other hospitals in the metropolitan area, both of the Insular Government and privately operated, together with their respective staffs have actively participated in our program.

NATIONAL RECOGNITION

The first Bulletin of the School of Medicine indicated that the curriculum for the training of our physicians was established in accordance with those offered by the leading medical schools in the United States, and conformed to the standards of the American Medical Association and the Association of American Medical Colleges. Annually throughout the four years of the evolution of our school representatives of these two great organizations visited our School, spent a period of nearly one week on each visitation, studying our program, discussing with the staff and administration and offering suggestions in the friendliest spirit of cooperation and finally sending a report of their findings. This assistance was of great benefit. In the September 12, 1953, issue of the Journal of the American Medical Association, the Council on Medical Education and Hospitals approved on May 29, 1953, the acceptance of our current seniors for internship in hospitals approved by the Council as if they had graduated from an approved medical school. This permitted them to apply to approved hospitals in the United States under the Matching Plan. On April 20, 1954, the culminating action occurred when the Executive Council of the Association of American Medical Colleges voted the University of Puerto Rico School of Medicine into full Affiliate Institutional Membership in the Association of American Medical Colleges. On April 28, 1954 the Council on Medical Education and Hospitals of the American Medical Association informed us that the School of Medicine of the University of Puerto Rico had been given full approval as a four-year medical school. It will be so listed in the next issue of the educational number of the Journal of the American Medical Association.

THE FUTURE

The title of this paper implies that only a beginning has been made. The University of Puerto Rico recognizes its educational responsibilities in the broad field of the Health Sciences. While a small beginning has been made in addition to the training of physicians as for example in Health Education, Public Health Nursing, Sanitary Science and Medical Technology, yet major fields remained untapped such as Dentistry, Collegiate Nursing programs

PLATE IV

Aerial view of the San Juan City Hospital, principal teaching hospital of the School of Medicine of Puerto Rico.

and other paramedical fields such as occupational therapy, physical therapy, X-Ray technology, hospital administration, etc. Postgraduate Medical education of a continuing nature as well as graduate work in the basic medical sciences are all pressing for attention. One area which cannot be neglected is that phase of postgraduate medical education relating to the internship and residency. With an alumnae group, half of whom will be in the Island next year as interns we will be constantly reminded that medical education is a process from which the school cannot extricate itself by the mere awarding of a degree.

SUMMARY

It is, indeed, a thrilling experience to be affiliated with a program such as the development of our Medical School in this era. It is another indication of the great progress Puerto Rico is making in educational, political, industrial, cultural and other fields. Congratulations to the dynamic leadership evidenced in our Commonwealth.

We of the School of Medicine, both faculty and student body, are cognizant of the responsibility imposed upon us by the manifest confidence in our future development. The economic support generously provided for our undertaking, the extensive cooperation already received and the recent national recognition of our standing makes it imperative that we strive even harder in the coming days to merit the confidence of our friends. The transition from a School of Medicine into a well rounded Medical Center will no doubt be a long and arduous process.

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APPENDIX

DEPARTMENT OF ANATOMY

NAME	TITLE
Carroll A. Pfeiffer	Professor of Anatomy and Head of the Department
José G. Frontera	Associate Professor of Anatomy
Luis R. Gumán López	Assistant Professor of Neuroanatomy and Neuropathology
Juan M. Bertrán	Assistant Professor of Applied Anatomy
Robert L. Dorsey, Jr.	Instructor in Anatomy

DEPARTMENT OF BIOCHEMISTRY AND NUTRITION

Conrado F. Asenjo	Professor of Biochemistry and Nutrition and Head of the Department
Marianne Goettsch	Associate Professor of Biochemistry and Nutrition
José A. Goyco Daubon	Research Associate in Biochemistry and Nutrition
Henry Jeffay	Associate in Biochemistry and Nutrition
Marta Cancio de England	Associate in Biochemistry and Nutrition
Elba Iris Porrata	Assistant in Biochemistry and Nutrition

DEPARTMENT OF MEDICINE

Rurico S. Díaz Rivera	Professor of Medicine and Head of the Department
Federico Hernández Morales	Professor of Clinical Medicine (Gastroenterology)
José N. Gándara	Clinical Professor of Medicine
Juan A. Pons	Clinical Professor of Medicine
Rafael Rodríguez-Molina	Clinical Professor of Medicine
Dwight Santiago-Stevenson	Clinical Professor of Medicine
Jaime Serra-Chavarry	Clinical Professor of Administrative Medicine
José A. de Jesús	Associate Professor of Medicine
Ernesto J. Marchand	Associate Professor of Clinical Medicine
Elí A. Ramírez	Associate Professor of Clinical Medicine
Angel Rodríguez Olleros	Associate Professor of Clinical Medicine
José Chaves-Estrada	Associate Clinical Professor of Administrative Medicine
David E. García	Associate Clinical Professor of Medicine
Eduardo Montilla	Associate Clinical Professor of Medicine
Eduardo R. Pons	Associate Clinical Professor of Medicine
José Rodríguez Pastor	Associate Clinical Professor of Medicine
Andrés E. Salazar	Associate Clinical Professor of Medicine
Ramón J. Sifre	Associate Clinical Professor of Medicine
Angel A. Cintrón Rivera	Assistant Professor of Therapeutics
Carlos E. Bertrán	Assistant Professor of Medicine
Agustín M. de Andino	Assistant Professor of Clinical Medicine
Federico Diez-Rivas	Assistant Professor of Clinical Medicine
José Torres Gómez	Assistant Professor of Clinical Medicine
Ramón A. Sifre	Assistant Professor of Clinical Medicine
Ernesto C. Martínez	Assistant Clinical Professor of Medicine
Enrique Pérez Santiago	Assistant Clinical Professor of Medicine
Bartolomé Borrás	Clinical Associate in Medicine

Name	Title
Roberto Busó	Clinical Associate in Medicine
Fernando L. Buxeda	Clinical Associate in Medicine
Eugenio Fernández-Cerra	Clinical Associate in Medicine
Jenaro Haddock	Clinical Associate in Medicine
Ulises López Sanabria	Clinical Associate in Medicine
Héctor Martínez-Villafañe	Clinical Associate in Medicine
Fernando M. Monserrate	Clinical Associate in Medicine
José Luis Porrata	Clinical Associate in Medicine
Julio V. Rivera	Clinical Associate in Medicine
Calixto Romero	Clinical Associate in Medicine
Ramón Suárez-Benítez	Clinical Associate in Medicine
Antonio Rivera Trujillo	Instructor in Medicine
Walter A. Cervoni	Instructor in Medicine (Clinical Pathology)
Francisco Ramos Morales	Instructor in Medicine
José Rullán	Instructor in Medicine
Raúl C. Vizcarrondo Vivas	Instructor in Clinical Medicine
José L. Robert	Clinical Instructor in Medicine
Juan Arruza	Clinical Instructor in Medicine
Pedro J. Durand	Clinical Instructor in Medicine
Juan Sabater	Clinical Instructor in Medicine
Antonio Marchany	Clinical Instructor in Medicine
Lilliane Ferrer Piñero	Clinical Instructor in Medicine
Francisco Trilla	Clinical Instructor in Medicine
Nelson Lugo Rigau	Clinical Instructor in Medicine
Jorge W. Mayoral	Assistant in Medicine
Juan A. Noguera	Assistant in Medicine
Mario R. García Palmieri	Assistant in Medicine
Héctor F. Rodríguez	Assistant in Medicine
Zoilo R. Sotomayor	Assistant in Medicine
René Silva	Assistant in Medicine
Manuel Pavía Fernández	Lecturer in Medicine
Abram S. Benenson	Lecturer in Medicine
Manuel Rodríguez-Ramos	Lecturer in Medical Jurisprudence
Federico Tilén	Lecturer in Medical Jurisprudence
Aaron B. Holman	Lecturer in Medical Jurisprudence

SECTION OF DERMATOLOGY

Víctor M. Rivera	Professor of Dermatology and Chief of Section
José F. Correa	Assistant Clinical Professor of Dermatology
Víctor J. Montilla	Assistant Clinical Professor of Dermatology
Jesús M. Quiñones	Assistant Clinical Professor of Dermatology

SECTION OF NEUROLOGY AND NEUROLOGICAL SURGERY

Luis R. Guzmán-López	Professor of Neurology and Neurological Surgery and Chief of Section
José Alvarez de Choudens	Assistant Professor of Clinical Neurology and Neurological Surgery
Nathan Rifkinson	Assistant Professor of Clinical Neurology and Neurological Surgery
Ricardo Cordero	Assistant Clinical Professor of Neurology and Neurological Surgery

SECTION OF PSYCHIATRY

NAME	TITLE
Juan Roselló	Associate Professor of Clinical Psychiatry and Chief of Section
Luis M. Morales	Clinical Professor of Psychiatry
Víctor Bernal	Assistant Clinical Professor of Psychiatry
Fernando Canino	Assistant Professor of Clinical Psychiatry
Ramón Fernández Marina	Assistant Clinical Professor of Psychiatry
Juan Homedes	Assistant Clinical Professor of Psychiatry
Juan E. Morales	Assistant Clinical Professor of Psychiatry
Ramón H. Señeri	Clinical Associate in Psychiatry
Jorge J. Dieppa	Assistant Clinical Psychologist
Mario Juliá	Lecturer in Psychiatry

SECTION OF ROENTGENOLOGY

José Landrón Becerra	Professor of Radiology and Chief of Section
Héctor M. Vallés	Associate Professor of Clinical Radiology
Laszlo Ehrlich	Associate Clinical Professor of Radiology
Carlos Guzmán Acosta	Assistant Clinical Professor of Radiology
Pablo L. Morales Rodríguez	Clinical Associate in Radiology
Rafael B. Díaz Bonnet	Clinical Associate in Radiology
J. Rivera Otero	Clinical Associate in Radiology
Juan R. Marchand Boneta	Clinical Associate in Radiology
Pedro Ramos Casellas	Lecturer in Radiology
Manuel Guzmán Rodríguez	Lecturer in Radiology
Carlos F. Jiménez Torres	Lecturer in Radiology

DEPARTMENT OF MICROBIOLOGY

E. Harold Hinman	Dean, School of Medicine, Professor of Microbiology and Head of the Dept.
José Oliver González	Associate Professor of Parasitology
Américo Pomales Lebrón	Associate Professor of Bacteriology
Irving Fox	Assistant Professor of Medical Entomology
José F. Maldonado	Assistant Professor of Parasitology
Rafael Mariñelarena	Assistant Professor of Bacteriology
Josefina Acosta Matienzo	Associate in Parasitology
Gladys Torres Lamoutte	Associate in Bacteriology
Carlos Fernández	Assistant in Bacteriology
María Dolores Pizarro	Assistant in Bacteriology
Juan E. Pérez Otero	Lecturer in Bacteriology

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY

Manuel Fernández Fuster	Professor of Obstetrics and Gynecology and Head of the Department
Jenaro Suárez	Professor of Clinical Gynecology
Rafael A. Gil	Associate Professor of Clinical Obstetrics and Gynecology
Jorge Héreter	Assistant Clinical Professor of Obstetrics and Gynecology
Miguel S. Dalmau	Assistant Clinical Professor of Obstetrics and Gynecology
José García García	Assistant Clinical Professor of Obstetrics and Gynecology

Name	Title
Angel R. Cestero	Assistant Clinical Professor of Obstetrics and Gynecology
Randolph McConnie	Assistant Clinical Professor of Obstetrics and Gynecology
Richard A. Gilbert	Assistant Clinical Professor of Obstetrics and Gynecology
Celso Ramón García	Associate in Obstetrics and Gynecology
José Díaz Carazo	Associate in Obstetrics and Gynecology
Edgardo Yordán	Instructor in Obstetrics and Gynecology
Thomas A. Cook, Jr.	Instructor in Obstetrics and Gynecology
Armando García Castillo	Clinical Instructor in Obstetrics and Gynecology
Rafael Vilar	Clinical Instructor in Obstetrics and Gynecology
Juan García Esteves	Assistant in Obstetrics and Gynecology
Manuel Fernández Durán	Assistant in Obstetrics and Gynecology
Paul Mari Rodríguez	Assistant in Obstetrics and Gynecology
Ramón Sánchez Viñas	Assistant in Obstetrics and Gynecology

DEPARTMENT OF PATHOLOGY

Enrique Koppisch	Professor of Pathology and Head of the Department
Félix M. Reyes	Associate Clinical Professor of Pathology
Mercedes Vicente de Torregrosa	Associate Professor of Clinical Pathology
Luis R. Guzmán López	Assistant Professor of Neuropathology
Raúl Marcial Rojas	Assistant Professor of Pathology
Francisco Lichtenberg	Assistant Professor of Pathology
Walter M. Bond	Assistant Professor of Pathology (on leave)
Lorenzo Galindo Merino	Associate in Pathology
Nathan Rifkinson	Associate in Neuropathology

DEPARTMENT OF PEDIATRICS

Antonio Ortiz	Professor of Pediatrics and Head of the Department
Egidio Colón Rivera	Associate Professor of Clinical Pediatrics
Dolores Méndez Cashion	Assistant Professor of Pediatrics
Juan Basora Defilló	Assistant Clinical Professor of Pediatrics
Ydalia Ortiz	Assistant Clinical Professor of Pediatrics
Dharma Vargas	Associate in Clinical Pediatrics
Enrique Milán	Clinical Associate in Pediatrics
Jenaro Scarano	Clinical Associate in Pediatrics
Miguel Firpi	Clinical Associate in Pediatrics
Etervina Figueroa	Instructor in Pediatrics
Osvaldo González	Instructor in Pediatrics
Mimosa Marín de Rullán	Instructor in Clinical Pediatrics
Eloísa Muñoz Dones	Instructor in Clinical Pediatrics
Héctor O. Hidalgo	Clinical Instructor in Pediatrics
José Sifontes	Clinical Instructor in Pediatrics
Aurea Muñoz	Assistant in Pediatrics
Katherine R. de Rivera	Assistant in Pediatrics
Hilda Martínez de Polo	Assistant in Pediatrics

DEPARTMENT OF PHYSIOLOGY AND PHARMACOLOGY

NAME	TITLE
Roger M. Reinecke	Professor of Physiology and Head of Department
David B. Tyler	Professor of Pharmacology
Carmen Busó de Casas	Associate Professor of Physiology
Frank E. South	Assistant Professor of Physiology

DEPARTMENT OF PREVENTIVE MEDICINE AND PUBLIC HEALTH

Guillermo Arbona	Prof. of Preventive Medicine and Public Health and Head of the Department
Juan A. Pons	Clinical Professor of Medicine
John W. Fertig	Visiting Professor of Biostatistics
Robert R. King, Jr.	Associate Professor of Preventive Medicine and Public Health
Félix M. Reyes	Associate Clinical Professor of Pathology
Mercedes V. de Torregrosa	Associate Professor of Clinical Pathology
Carmen Acevedo de Torres	Assistant Professor of Health Education
Nelson Biaggi	Assistant Professor of Sanitation
Celia Guzmán	Assistant Professor of Public Health Nursing
Rafael Timothée	Assitant Professor of Public Health Practice
Rafael Vilar	Assitant Professor of Public Health Practice
Orlando Bonilla Soto	Assistant Professor of Bacteriology
Juan Basora Defilló	Assistant Clinical Professor of Pediatrics
Félix Astacio	Instructor in Sanitary Science
Francisco Berio	Instructor in Public Health Practice
María M. García	Instructor in Public Health Nursing
Francisco Mejías	Instructor in Medical Technology
Edris Rice-Wray	Instructor in Public Health Practice
Adelaida Sanavitis	Instructor in Public Health Nursing
Angel M. Cabrera	Assistant in Medical Technology
Victoria Smith Ramos	Assistant in Public Health Practice
Borinquen Mussenden	Assistant in Public Health Practice
Gregoria Auffant	Lecturer in Public Health Nursing
Jennie Báez de Kalil	Lecturer in Public Health Nursing
Adelina Burgos	Lecturer in Public Health Nursing
Catalina Burgos de Maymí	Lecturer in Public Health Nursing
Angeles Cebollero	Lecturer in Public Health Education
Aida Colón de Ferrer	Lecturer in Public Health Nursing
Angeles Díaz	Lecturer in Public Health Nursing
Dolores García de Martínez	Lecturer in Public Health Nursing
Félix García	Lecturer in Sanitary Science
Nicolina Pabón	Lecturer in Public Health Nursing
Isabel Pérez de Hernández	Lecturer in Public Health Nursing
Belén Ramos de Méndez	Lecturer in Public Health Nursing
Angela Díaz de Gutiérrez	Lecturer in Public Health Nursing
José L. Janer	Lecturer in Public Health Practice
Lágrima Marín de Márquez	Lecturer in Public Health Education
Margarita Matttei de Méndez	Lecturer in Public Health Nursing
Rafaela Salgado de Rosado	Lecturer in Public Health Nursing

Name	Title
Enrique Toro	Lesturer in Sanitary Science
María Zalduondo	Lecturer in Public Health Education
Cecilia Fonseca	Lecturer in Public Health Nursing

DEPARTMENT OF SURGERY

José Noya Benítez	Professor of Surgery and Head of the Department
Luis A. Passalacqua	Clinical Professor of Surgery
Francisco L. Raffucci	Associate Professor of Surgery
Roberto J. Jiménez	Associate Professor of Clinical Surgery
Gumersindo Blanco	Assistant Professor of Surgery
Manuel Baralt	Assistant Clinical Professor of Surgery
Luis A. Díaz Bonnet	Assistant Professor of Clinical Surgery
Alberto Adam	Assistant Clinical Professor of Surgery
Salvador Busquets	Assistant Clinical Professor of Surgery
Angel S. Casanova Díaz	Assistant Clinical Professor of Surgery
Marcos Dones	Assistant Clinical Professor of Odontology
Blas Ferraiouli	Assistant Clinical Professor of Surgery
Herman Flax	Assistant Clinical Professor of Physical Medicine and Rehabilitation
J. R. González Giusti	Assistant Clinical Professor of Surgery
Alfred L. Axtmayer	Assistant Clinical Professor of Surgery
Jaime F. Pou	Assistant Clinical Professor of Surgery
Dávid Rodríguez Pérez	Assistant Clinical Professor of Surgery
José S. Licha	Assistant Clinical Professor of Surgery
Pedro Suau	Assistant Clinical Professor of Surgery
Juan M. Bertrán	Clinical Associate in Surgery
Angeles Díaz	Clinical Associate in Surgery
William P. Gelpí	Clinical Associate in Surgery
José A. Sárraga	Clinical Associate in Surgery
Herbert Mayer	Clinical Associate in Surgery
Héctor M. Nadal	Clinical Associate in Surgery
Antonio Ramos Oller	Clinical Associate in Surgery
Walter Benavent	Clinical Instructor in Surgery
Marvin S. Cashion	Clinical Instructor in Surgery
E. de Hostos	Clinical Instructor in Surgery
Félix Rodríguez Forteza	Clinical Instructor in Surgery
Pedro Rullán	Clinical Instructor in Surgery
Lawrence J. Snyder	Clinical Instructor in Surgery
Mario Tomasini	Clinical Instructor in Surgery
Alvaro Alfonso	Assistant in Surgery
Raúl Armstrong Mayoral	Assistant in Surgery (military leave)
Taufick Bendeck	Assistant in Surgery
Dorothea Weybright	Assistant in Surgery
José M. Blanco	Assistant in Surgery (military leave)
John F. Sanabria	Assistant in Surgery
Manuel Astor	Lecturer in Surgery
Guillermo Barbosa	Lecturer in Surgery
Basilio Dávila	Lecturer in Surgery
A. Oliveras Guerra	Lecturer in Surgery

SECTION OF ANESTHESIOLOGY

NAME	TITLE
Frederick J. González	Professor of Anesthesiology and Chief of Section
[illegible]to Colón Yordán	Clinical Associate in Anesthesiology
[illegible]uel Figueroa	Clinical Instructor in Anesthesiology
Iván H. García	Assistant in Anesthesiology
Ariel Méndez	Assistant in Anesthesiology

SECTION OF NEUROLOGICAL SURGERY

Luis R. Guzmán	Professor of Neurology and neurological Surgery and Chief of Section
José A. Alvarez de Choudens	Assistant Professor of Clinical Neurology and Neurological Surgery
Nathan Rifkinson	Assistant Professor of Clinical Neurology and Neurological Surgery
Ricardo Cordero	Assistant Clinical Professor of Neurology and Neurological Surgery

SECTION OF OPHTHALMOLOGY

Guillermo Picó	Professor of Ophthalmology and Chief of Section
Antonio Navas Torres	Clinical Professor of Ophthalmology
Roberto Buxeda	Assistant Clinical Professor of Ophthalmology
José A. Gallardo Díaz	Assistant Clinical Professor of Ophthalmology
Nicolás Quiñones Jiménez, Jr.	Clinical Associate in Ophthalmology
Andrés Montalvo Carroll	Clinical Associate in Ophthalmology
Rafael Maldonado	Clinical Instructor in Ophthalmology
Lydia Pérez Guardiola	Clinical Instructor in Ophthalmology

SECTION OF ORTHOPEDIC AND FRACTURE SURGERY

Peter E. Sabatelle	Professor of Orthopedic and Fracture Surgery and Chief of Section
Ian Douglas Murphy	Clinical Instructor in Orthopedic and Fracture Surgery
Aníbal Lugo	Clinical Instructor in Orthopedic and Fracture Surgery
Leon Sheplan	Lecturer in Orthopedic and Fracture Surgery

SECTION OF OTOLARYNGOLOGY

José Picó	Professor of Otolaryngology and Chief of Section
Carlos Muñoz MacCormick	Professor of Clinical Otolaryngology
Juan H. Font	Clinical Professor of Otolaryngology
Miguel Alonso	Clinical Professor of Otolaryngology
William Reichard	Assistant Clinical Professor of Otolaryngology

NAME	TITLE
Lorenzo Arsuaga	Clinical Associate in Otolaryngology
Jaime H. Font	Clinical Associate in Otolaryngology
Frank Quiñones Jiménez	Clinical Associate in Otolaryngology
Antonio Rullán	Clinical Associate in Otolaryngology
Nicolás Quiñones Jiménez	Lecturer in Otolaryngology

SECTION OF UROLOGY

Luis Sanjurjo	Professor of Urology and Chief of Section
Pablo G. Curbelo	Clinical Professor of Urology
Alberto Mejía Casals	Associate Clinical Professor of Urology
Luis M. Isales	Clinical Associate in Urology
Néstor Méndez	Clinical Instructor in Urology
Benigno Rodríguez Lucca	Clinical Assistant in Urology
Esteban García Cabrera	Lecturer in Urology

SELECTION OF STUDENTS AT THE SCHOOL OF MEDICINE SCHOOL OF TROPICAL MEDICINE OF THE UNIVERSITY OF PUERTO RICO

CARROLL A. PFEIFFER*

It is pretty well agreed that the selection of students is one of the more important prerequisites for the successful operation of a medical school. Certain aspects of this procedure are well standardized among the medical schools of the United States and have served as a pattern for the selection of students here, since it was desirable, in view of the relationship of Puerto Rico to the United States, that this school be recognized by the American Medical Association and the Association of American Medical Colleges. This recognition has recently been granted.

The mechanism of selection. The first class was selected directly through the chancellor's office. The second class was chosen by a Committee on Admissions who began to evolve detailed application forms and a comprehensive confidential questionnaire to take the place of letters of recommendation. The Committee on Admissions which consists of seven members, is appointed by the Dean who also acts as an ex-officio member of the committee but does not vote. This committee is composed of about equal numbers from the basic science and clinical faculties.

Requests for information are sent to the dean's office. If it seems unlikely that the candidate can qualify, he is notified by letter of our special requirements, which gives him a chance to reconsider whether it is worth while for him to make application to our school. This eliminates a certain number of potential applicants from the States without the expense of sending out the complete application materials. These consist of a copy of the Bulletin of the School of Medicine, two copies of the application blank, three confidential questionnaires, two forms for requesting transcripts of credits, and six printed envelopes addressed to the Office of the Dean. The closing date for applications is October 31, and the first year class is selected by January 1. All applicants are notified of the decision of the committee at this time.

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* Chairman of the committee on Admissions and Head of the Department of Anatomy, School of Medicine School of Tropical Medicine University of Puerto Rico.

To the hard working members of the committee, Drs. J. A. De Jesus, M. Fernández Fuster, J. G. Frontera, R. R. King, A. Pomales Lebrón and F. L. Raffucci, belong all of the credit for its successful operation and for the selection of our medical classes.

The chairman assumes all responsibility for any errors of interpretation of the function of this committee as set forth in this discussion.

Procedure. The applicant fills out the application forms, sends one to the Office of the Dean and keeps the other for his own file. An essay of 200 to 300 words, written by hand in English, must accompany this form. The applicant is responsible for seeing that the dean's office receives an official transcript of all of his college work, and two letters of recommendation, one preferably from the premedical committee of the college, if it has one. He is required to take the Medical College Admission Test and must have his score forwarded to the dean's office. The application is checked for completeness and accuracy of figuring grade point averages by a secretary in the dean's office. The folder containing all of the above material is numbered and is then turned over to the chairman of the admissions committee who sees that each member of the committee examines each application and fills in a summary sheet which contains all of the pertinent information on the candidate. The summary sheet is set up so that there are five qualifications which can be rated separately. These are: (1) medical aptitude test, (2) general factors such as age, extra curricular activities and physical and mental defects, (3) scholastic record, (4) attitude as expressed by the written essay and in the letters of recommendation and (5) personal interview. Each committee member determines individually how much weight he will give to each of the categories in order to arrive at his final rating of the candidates.

After all applications are seen by each member of the committee, a meeting is held to determine which candidates are worthy of being interviewed. The candidates are separated into three groups: (1) those to be interviewed, (2) those to be placed in reserve, and (3) those who are readily rejected. Any member of the committee can, by his vote alone, place a candidate in the group to be interviewed or to be reserved even though all other votes are for a lower category. This might seem to increase the number of candidates to be interviewed out of proportion to their records, but has not worked out to be any burden on the committee and it does allow the committee as a whole to determine whether some special attribute that any one member sees in a candidate is sufficiently worthy to allow his being accepted. The dean, as an ex-officio member of the committee, discusses the candidates but does not vote.

The chairman of the committee then arranges for interviews with all of the candidates in the first group. All members of the committee and the dean, as a group, interview these candidates with the view of evaluating personal appearance, poise, personality, interest in medicine, keenness of intellect, and ability to think under tension. Each member scores the candidates on a scale of

1 to 4, and the scores are averaged to arrive at a committee score. Each member records his individual score and the committee score on his summary sheet. If there are too many candidates from the first group who do not show up well in the interviews, then the better candidates from the reserve group may be interviewed.

After the interviews each member of the committee rates the candidates in the order of his preference from 1 to the number which corresponds to the total number of applicants interviewed, using all of the criteria on his summary sheet. At the meeting held to select the first year class of 52 students, the Puerto Rican boys, Puerto Rican girls, and non-Puerto Ricans are chosen separately. However, the number of girls selected, 6 to 8, depends on their relative standing in the over-all rating of the candidates. Usually only one or two non-Puerto Rican boys are chosen. In selecting the Puerto Rican boys any candidate that is rated below 40 by each member is automatically accepted. Any candidate rated beyond 40 by any member must have a committee rating which is arrived at by taking the sum of the ratings of all of the members of the committee. After the committee ratings are obtained, candidates with the lowest total scores are accepted in the order of their ratings until there are the required number of names on the accepted list. The candidates having the next 8 lowest committee ratings are named as alternates. An alternate is also chosen for the Puerto Rican girls and one for the non-Puerto Rican boys, making a total of 10 alternates.

Requirements. While the minimum requirements set up by the School of Medicine of the University of Puerto Rico have much in common with those of the accredited schools of medicine in the United States, there are certain requirements, such as language, that are peculiar to our environment. We require a minimum of three years of work (90 semester hours) in an approved college, which must include 16 semester hours of chemistry including an approved course in organic chemistry, 12 semester hours each of Spanish and English composition and literature and 8 semester hours each of physics and biology. We prefer that the applicant has the bachelor's degree and recommend that he obtain as well rounded an education as possible.[1] It is axiomatic that a Doctor of Medicine assumes a place of influence and social leadership in his community. If he is to fulfill properly this obligation, he needs a cultural background, which is often best obtained by a broad liberal education in college. His preclinical courses will be essentially scientific in nature, and, because of the rigorous study required during his training in medicine, he will find little time during this period to improve his mind in the broader aspects of learning. Each candidate must take the Medical College Admis-

sion Test, preferably in May, but not later than November of the year before he wishes to enter medical school. He must request that the testing service send to the dean's office his scores on this test.

Our minimum requirements may tend to be confusing to the premedical committees of the colleges, who feel, and probably with some justification, that we would not take a candidate who fulfills only these requirements, particularly only the 8 semester hours of biology. Our answer is that we would take him if he were a good student and could convince us that he had a deep and earnest desire to learn and practice medicine. However, this situation rarely arises because a deep interest in medicine can scarcely exist without sufficient interest in biology to cause the candidate to take more than the minimum number of hours in this subject. In our opinion it is much more important what he takes in biology than how little he can take. We do not feel that we should dictate what biology courses he should take, but he should have as well rounded an understanding of this science as it is possible to obtain within the limits of the number of courses he takes. Any course well taught and interestingly given is preferable to any prescribed course not interestingly given but presented because it is required. In any science we do not want him to take a subject that will be repeated in medical school. This immediately brings up the question of embryology. Certain medical schools expect that their students will have had all of their embryology before they get to medical school. We, on the other hand, teach a course in human embryology in the Department of Anatomy and would prefer that our students had not studied this subject as undergraduates since our interpretation of this material must, of necessity, be much different from that given in a non-medical course. It often requires more time for the student to make the shift in emphasis than it does to master the material for the first time. An introductory course in comparative vertebrate embryology properly given to cover the fundamentals of embryology, would be very helpful. However, only a small proportion of the premedical students in any one college come to us and less than half of the students who apply to medical schools are admitted. For the latter and for those students who go to the schools that require embryology before they are admitted, a course in human embryology at the undergraduate level is indicated. You see how complicated the discussion of a single course can be. The ultimate decisions must rest with the premedical committees, which shows the need for close liaison between the premedical and admissions committees (to be discussed later). A good case can be made for a course in comparative anatomy, introductory or comparative physiology and histological

technique, but probably an even better case can be made for such courses as genetics or organic evolution.

As far as chemistry is concerned, we probably need to be a little more specific in our requirements since the student is less liable to realize the need for such a good grounding in this subject. In addition to organic chemistry, we would even go so far as to recommend that quantitative and qualitative analysis should be taken. Also, even though we are in the atomic age, it is necessary to have the requirement of 8 hours of physics to be certain that our candidates are prepared in this subject.

Our requirement of 12 semester hours each of English and Spanish are unique for this school. The reason for it is obvious. All instruction is in English, but the clinical patient will speak only Spanish. Therefore, the real requirement is that the student must be able to converse in both languages. Our interpretation of this requirement is, therefore, subject to a great deal of variation. It is evident to us that a Puerto Rican student going to college in the States will probably not be allowed to take Spanish for credit as that is his native tongue and, therefore, he will not be able to show any college credits in Spanish. If his native tongue is Spanish he will be able to converse with the patient and if he does well in college in the States his English is adequate also. If a Puerto Rican is educated in Puerto Rico our primary concern is that he can handle the English language. An extended period in the States where he is forced to use English is, of course, a much more convincing argument for this adequacy than only credit hours in college. The realization of the requirement of a true speaking knowledge of Spanish has apparently very effectively limited the number of applications from poor students in the United States.

The requirement that the student take the Medical College Admission Test is somewhat of an experiment, but we are convinced that the scores are very helpful criteria. Manifestly, the social and cultural background in Puerto Rico is different from that in the United States, for whose students this test is designed. It is also evident that the scores of Puerto Rican students educated in the States would be different from those completely educated in Puerto Rico. We do not yet have enough material to analyze this information adequately.

Who should apply? Theoretically, anyone who wishes may make application to enter the School of Medicine of the University of Puerto Rico, yet it is obvious that unless he can meet our minimum requirements, he is wasting his effort to apply. There are, however, many students who will have the minimum, or can obtain the minimum requirements as printed in our catalog, who, be-

cause of their scholastic record or other reasons would stand no chance of admission to an accredited school of medicine. These students should be discouraged from applying because they waste not only their own time but also the time of our committee, as well as of any persons asked to write recommendations. Many schools require a non-refundable money deposit in order to discourage applicants in this group. If the applicant is accepted, the money applies toward his fees. We do not wish to use any means of this kind to discourage applications because many of our worthy applicants would find it extremely difficult to comply with such a requirement. Often, a student that we accept must borrow the $25.00 deposit which must be made to guarantee that a place will be held for him in the entering class. In Puerto Rico we depend on the premedical committees of the various schools to discourage the students who obviously have no chance to be accepted. This has been very effective in practice. However, at the same time, we do not want these committees to be so critical that they recommend that only those students apply whom they are confident will be accepted, because a student who might otherwise be able to prove to us his ability to study medicine might be denied this right. We do not require that an applicant have a letter of recommendation from the premedical committee of his school as it is conceivable that an applicant honestly feels that others of his professors may have a much better chance to evaluate his abilities. If the applicant makes this choice it is not held against him, but if he is given a personal interview, he is always asked to explain why he did not request a letter of recommendation from the premedical committee.

With non-Puerto Rican students, the language requirement, as mentioned above, is quite effective in limiting the number of applicants from among poor students in the continental United States. These students are not very likely to be proficient in Spanish.

The number of students who apply. In the beginning, the unusual conditions following the end of the war caused a piling up of a large backlog of students in Puerto Rico, some very good ones, who were unable to get into medical schools in the United States. These, and the usual number of borderline students who always apply to a new school of medicine, made the number of applications well over 300, from which 50 students were selected. With the disappearance of this backlog, the understanding of the need for good scholarship and the extremely good liaison between our committee and the premedical committees on the Island have now limited the number of applicants to about 125, of whom we now choose 52.

Breakdown of the students applying and accepted. Disregarding the unusual situation which existed at the beginning, the pattern of distribution of students applying and accepted is shown in table 1. It may readily be seen that most of the students applying

TABLE I

BREAKDOWN OF THE APPLICATIONS STUDIED FOR THE CLASSES ENTERING IN 1953 AND 1954

Classification	*No. applying*		*No. interviewed*		*No. accepted*	
	1953	1954	1953	1954	1953	1954
Puerto Rican boys U.S. trained	15	18	6	11	4	9
Puerto Rican boys locally trained	60	74	51	50	36	35
Non Puerto Rican boys	17	9	3	4	2	1
Puerto Rican girls U.S. trained	2	7	1	1	0	0
Puerto Rican girls locally trained	17	14	11	12	8	7
Non Puerto Rican girls	3	3	0	0	0	0
Total	116	126	72	78	50	52
Total Puerto Rican boys	75	92	57	61	40	44
Total Puerto Rican girls	19	21	12	13	8	7

are Puerto Ricans and that we accept almost entirely Puerto Rican students. The non-Puerto Rican boys accepted usually have some special connection to the medical problems of Puerto Rico or a similar area. It may also be seen that we accept somewhat less than one-half of the Puerto Rican boys who apply and about one-third of the Puerto Rican girls. The percentage accepted[2] of the students who apply is approximately that of the average for the United States. However, the ratio of girls to boys accepted here (16 to 20%) is much higher than in the United States (5%).[2]

Why do we take more women than other schools? Except for Women's Medical College of Pennsylvania, which takes only women students, we accept a higher proportion of women than any other school. In the United States, as here, the number taken is not dependent on their relative academic records. It is readily conceded by everyone that a woman must have a much better record than a man to be admitted into medicine. This is because the number of years in practice by women is much below that of men. Therefore, the admissions committees attempt to limit, as much as possible, the factor of academic failure of women in medicine. The factor of matrimony is an unknown. The present concept of marriage

and a career for women has, to a great extent, prevented women students from dropping out of school, when marriage occurs during their training period. It has also somewhat lengthened the number of years of practice for women physicians and will probably continue to do so. There is very little outlet in Puerto Rico for women with an inclination toward medicine if they are not accepted in a medical school, and, therefore, their incentive is exceptionally high. Taking all factors into consideration, we find it very difficult to set an arbitrary figure lower than the number we take. Admitting this number of women students is somewhat of an experiment, but most of the members of the committee feel that with the health problem what it is in Puerto Rico, this is a pretty good risk. We have never yet taken a non-Puerto Rican girl, because we have had too many good local girls.

Intangibles in the selection of students. It should be obvious to everyone that there are many intangibles in the selection of students. Sometimes these have to be the deciding factor of whether a candidate is selected or rejected. The committee admits that the intangible factors are often personal impressions, but while the committee may make errors, it always acts with honest convictions. There are some things that can be fairly readily explained, such as the influence of the age of the candidate. If the average age of the applicants who enter medical school is 22, it is quite obvious that, other things being equal, a candidate 32 years old will have ten years less of active practice. We have no age limit, but a candidate over 30 years of age must have a more convincing reason for obtaining the medical degree than if he were near the average age. Even the question of grades allows for considerable interpretation. One student works hard to make a C+ or B — average. Another student makes the same average with no effort at all. Will the first student be able to maintain passing standards in medical school? Will the incentive of a medical education make a good medical student out of the second? It is also evident that a B grade in one college is a different measure of ability than the same grade in another college. To anyone with an extended experience in teaching in medical school, it is obvious that certain students that maintain with difficulty a C average in medical school may make better doctors than many straight A students.

Our committee has one decision to make that is peculiar to this school. It often has to decide whether the candidate's ability in English is such that it will not fatally hamper his performance in the pre-clinical years. We believe that our efforts to have this condition remedied at the undergraduate level are being effective, and we hope that it will soon not be a factor in our decisions.

Lastly, there are moral factors. What makes a candidate morally unfit to be a physician? On the surface it would seem that the factors involved would be as clear as black and white, but unfortunately the distinctions may not be that evident. Instead, one often has to evaluate varying degrees of gray.

Relations with the premedical committees. Only about 15% of the students accepted are trained in the United States. The rest come from the Island. More than 60% come from the campus of the University of Puerto Rico in Río Piedras. This gives us an unusually good chance to work closely with the premedical committees of the schools that supply most of our students. We have at least two joint meetings a year with the premedical committee at Río Piedras, and several of our members have had friendly contacts with the committees of the other schools on the Island. We feel that we have very good liaison with the premedical committees and are convinced that this is paying dividends in that they are now sending us candidates that we feel much more nearly approach what we would like to have. We feel that this trend will continue. We do not expect the premedical committees to do our work of selecting students, but they can most effectively interpret to the premedical student what we would like and can see that the student fulfills these qualifications to the best of his ability.

Who should the candidate ask to write letters of recommendation? It is evident that the person writing a letter of recommendation should know as much as possible about the student he is recommending. It is also evident that two persons who know the candidate equally well may evaluate him differently, but even though they make the same evaluation, they might very well write letters that would be interpreted entirely differently. Therefore, we have developed a factual type of confidential questionnaire to be used in place of the ordinary letter of recommendation. Even so, it is helpful to us when we are acquainted with the type of recommendation an individual gives. We learn to evaluate the responses of the premedical committees to the questions we ask. This, with the fact that the premedical committees may know more of what we want, is why we prefer that the candidate always has a recommendation from them. We prefer that the premedical committee give us a composite evaluation of the candidate and that any variations of opinions among its members be fully and freely presented. It may be that the premedical committee can effectively devise a form that more readily lends itself to the expression of their committee action. We would gladly accept this in place of our own. Any other recommendation should probably come from some member of the faculty under whom the applicant has done considerable work.

What outlet is there for the premedical student who fails to be accepted in an accredited medical school? We accept less than half of the students who apply for admission. A few of those that we do not accept may be admitted to other accredited schools. However, the situation here is such that this happens relatively rarely. Candidates whom we accept may also be accepted elsewhere and sometimes choose the other school instead of ours. What can the other students do? They may go to numerous foreign medical schools which are unrecognized by the American Medical Association. We do not recommend their enrolling in these schools. If they do not, what can they do? There is little outlet for them with their college training. We have no graduate, dental or veterinarian school. Physical therapy and medical technology take a few. Most of the others must adjust to some entirely different occupation. A full discussion of this problem is not in the scope of this paper, but it is one that needs attention.

SUMMARY

The mechanism by which students are selected for the School of Medicine School of Tropical Medicine of the University of Puerto Rico is described, and an attempt is made to point out some of the difficulties encountered in this selection and how these difficulties are being overcome. The minimum requirements for admission are discussed, and those portions of them most questioned are explained from our point of view. More than 60% of our students come from the Río Piedras campus of the University of Puerto Rico, and only 15% are trained in the United States. The rest come from the remaining colleges on the Island. Therefore, liaison between the Committee on Admissions and the premedical committees of the colleges that supply us with most of our students is readily possible and very effective.

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THE DEVELOPMENT OF TEACHING AND RESEARCH IN PHYSIOLOGY

ROGER M. REINECKE*

The Department of Physiology and Pharmacology was one of the new basic science departments of the School of Medicine in the sense that it was not developed on the basis of a preceding department in the School of Tropical Medicine. This account will deal only with the development of teaching and research in physiology. The new department came into being as such early in July of 1950 when I arrived in Puerto Rico to plan and organize the details of its practical operation. Dr. Carmen B. Casas joined me at that time from the Department of Medicine of the School of Tropical Medicine where she had been working while waiting for the new department to be organized and to start to actually function. The remaining months in 1950 were devoted to the development of the facilities and the obtaining of the essential apparatus and supplies required to teach the laboratory part of the course in physiology for the first year students in medicine which was scheduled to start early in January 1951.

The department was fortunate in securing the full-time services of Dr. Rodney B. Harvey who joined us in December of 1950 so that a full-time staff of three were available to teach the course in physiology for the first time. Two physicians from the community, Dr. N. Rifkinson and Dr. F. J. González also generously contributed a number of lectures in areas of physiology related to their clinical specialties. The course was taught without serious difficulty although some facilities were not fully completed when it started and some items of apparatus were still missing and therefore had to be improvised.

In the three subsequent years in which the course has been taught, it has been progressively improved by the introduction of new material, especially in the teaching laboratory. In 1952, Dr. Harvey initiated the use of modern electronic recording equipment and strain-gauges in the student laboratory. This program was extended in 1953 and 1954 so that in the current year, 1954, the students have had the opportunity to record electroencephalograms and intracardiac pressures as a regular part of their laboratory work. In the current year a series of laboratory experiments also have been initiated in which the students have undertaken the measurement of the body fluids such as plasma, extracellular fluid, and total body water by the dilution technic.

The department of Surgery has cooperated by instructing the

* Professor of Physiology and Head of the Department.

students in physiology in the technics of experimental surgery during the later periods which were scheduled for physiology laboratory. This has been an interesting experiment in interdepartmental cooperation and has provided the students with valuable experience. Dr. Frank Raffucci, of the Department of Surgery has made the outstanding contribution to the success of this experiment.

Arrangements have also been made to take the class in physiology from time to time by bus to one or another of the hospitals in the San Juan area where physicians on the hospital staff would then present patients to the class who exemplified some aspect of the application of physiological knowledge to the diagnosis and treatment of disease. The experiment on a whole seems to have been successful in furthering the development of insight on the part of the students into the relation between their work in physiology and their later studies in clinical subjects.

Dr. Richard R. Bob filled the position vacated by Dr. Harvey, when he returned to the continental United States at the end of the 1951-52 school year, on temporary basis during the presentation of the course in 1953. Dr. Frank E. South, Jr. joined the department in August of 1953 to complete our regular full-time staff of three in physiology.

The members of the teaching staff in physiology have carried on vigorous research programs in order to maintain and extend their scholarly competence as physiologists. Dr. Casas and I have been successful in obtaining grants from outside the school to support our research programs. Dr. Casas has received a total of $25,068 for this purpose and I $17,600 since the department was organized. The medical school supported Dr. Harvey's research while he was with the department and has provided the means for Dr. South to get his research program started.

Dr. Casas has continued her researches into the relations between the thyroid and adrenal which she had initiated while a member of the Department of Medicine of the School of Tropical Medicine. She has demonstrated that the adrenal tumors which appear spontaneously in some strains of imbred mice after castration develop even though the function of the thyroid has been depressed but that they do not secrete estrogen as they do when thyroid function is normal. She has also observed the formation of liver tumors in these mice when they have been given thyroid depressing drugs such as thiourea or thiouracil for a prolonged period. Dr. Casa's studies are more fully described in the following papers:

1. King, J. T., C. B. Casas, M.B. Visscher, The estrus behavior and mammary cancer incidence in ovariectomized C3H mice in relation to calorie intake. Cancer Res. 11:712, 1951.

2. Casas, C. B., Adrenal glands of the C3H mouse castrated after prolonged treatment with thiouracil. Am. J. Physiol. 171:713, 1952.

3. Casas, C. B., E. Koppisch, The thyroid and adrenal glands of castrated C3H treated with thiourea. Endocrinology, 51:322, 1952.

4. Casas, C. B., Response to exogenous estrogen of ovariectomized C3H mice treated chronically with thiouracil. Fed. Proc. 13:23, 1954.

Some of the studies on the local effects of ischemia with which I had been engaged just before coming to Puerto Rico have been completed. These have shown: 1. how long a period of ischemia is necessary to disrupt the mechanisms which keep a region of the body at its usual volume, i.e, prevent the accumulations of tissue fluids which lead to swelling and edema, 2. some of the effects of ischemia on the subsequent circulation within the region which had been subjected to ischemia and the region immediately proximal. For further details see:

1. Nudell, G. and R. M. Reinecke, Am. J. Physiol. 168:189, 1952.

2. Edwards, C. and R. M. Reinecke, Am. J. Physiol. 174:289, 1953.

It has also been possible for me to extend my investigations on the role of the kidney in organic metabolism. It was found that the kidney of the eviscerated monkey contributes sugar to the blood flowing through it. For the details of this study see:

Reinecke, R. M., Am. J. Physiol. 171:29, 1952.

In subsequent unpublished studies it also has been found that at times the kidney appears to remove lactate from the blood which cannot be accounted for by excretion in the urine. Further evidence has also been obtained concerning the role of the kidney in the metabolism of fructose, but the studies in this area have not been completed.

Dr. South had been interested in the effects of the "inert" gases such as nitrogen, helium, argon, etc. on the metabolism of isolated tissues before joining the department. He has continued in his interest in this field and has initiated a series of researches in it which he is just getting underway.

PRESIDENTIAL ADDRESS - THIRD ANNUAL MEETING, PUERTO RICO CHAPTER OF THE AMERICAN COLLEGE OF SURGEONS

JOSE NOYA-BENITEZ, M.D., F.A.C.S.

In the few minutes that I have left of the time that was assigned to the Presidential address I have been asked by one of my favorite associates to talk on something which is not very scientific and certainly not startling: "On the behavior of the surgeon on the Operating Room". He was moved to do this by reading the introduction of Victor Bonney in his book on Gynecologic Surgery. Last year I intended to do so but the fact that we were starting on our clinical teaching of surgery at our new Medical School made me change my subject to cover our aims and purposes in the teaching of surgery to medical students.

"The pompous repetition of the obvious" to quote a phrase I heard last year from Dr. Grantley W. Taylor, is something which is found too frequently, and something which by all means I would try to avoid in front of this distinguished audience. I hope by all means not to be pompous, and almost as hard, I will try not to be repetitious. I will quote freely from Sir W. H. Ogilviey article on The Gentle Surgeon in the Lancet, 1936, pp. 435-36.

"ON THE BEHAVIOR OF THE SURGEON IN THE OPERATING ROOM"

In Guy de Chauliac's theme on the conditions necessary for a surgeon he stated:

First: He should be learned

Second: He should be expert

Third: He should be ingenious

Fourth: He should be able to adapt himself

It is with the fourth requisite, and especially that part of it applied to behavior in the operating room, that we are concerned. This presuposes of course, that the surgeon fulfills the first three requisites.

"Let the surgeon be bold all sure things, and fearful in dangerous things, let him avoid all faulty treatments and practices. He ought to be gracious to the sick, considerate to his associates, courteous in his prognostications. Let him be modest, dignified, gentle, pitful, and merciful."

Let him be humble, for he should remember Ambroise Paré words: "I dressed his wound, God healed him". And let the surgeon remember the great physiological truth so beautifully enunciated by John Hilton: "Nature has a constant tendency to re pair the injuries be the result of fatigue or exhaustion, of inflammation or accident".

The surgeon's aim should be to help nature repair or circumvent the damage it has suffered, or how to best initiate the normal function if repair is impossible.

Above all things let the surgeon be gentle. Gentle handling of the tissues should be accompanied by gentle handling of his colleagues and associates, from his first assistant to his scrub nurse, the anesthetist, and all the persons that form the operating room team, including the O. R. Supervisor. "The tales of surgeons who were uncouth and choleric, of retorts whose discourtesy made them classic, of bold and skillful but brutal manipulations, of blood and agony, of boastful self-assertion, etc. etc., were perhaps excusable in the days when poor and dangerous anesthesia made speed necessary, but not now."

It is true that asepsis, better and safer anesthesia, better preparation of the patient have made more extensive operations possible, and the need if not for speed, for not delaying (or dilly-dallying as Dr. A. Whipple would say) remains, but not at the risk of slashing the way through tissues.

"It is true that human nature and specially adolescent human nature as examplified by the medical student, sometimes will prefer the spectacular to the artistic, and flock where the blood flows more freely, where the shouts are the loudest, where instruments are thrown about the theatre, where "look and see", the infallible solvent of diagnostic difficulties, ensures an abundant succession of "tours de force". Yet this same medical students when they become residents and fellows in surgery, will come to the gentle surgeon to learn the details for tissue kindness which are unseen from the gallery.

The criteria of a great surgeon in the operating theatre is that the tissues shall be treated with the greatest gentleness, subject to the least damage, replaced carefully, apposed accurately, in short, that they shall bear the least trace of the surgeon's passage.

The surgeon should be sure before starting on his procedure that he has all the instruments necessary, all the sutures that will be needed and adequate help. If it happens that they are not available when he needs them, it is nobody's fault but his and he should not blame acrimoniously, anybody in the operating room but devise means of accomplishing the operation with the means at hand. Any severe reprimand in the operating theatre would only lower the efficiency and might be, the cooperation of the operating team, and will deter from the perfection that we should try to atain in all operations.

We are all humans. Pompous, perhaps not, repetitious, obviously, and can not expect the surgeon not to show some emotional reaction when things are not going well. But this is just the time to

show the surgeon's mettle and when ability and gentleness may obtain the best results from a difficult situation.

Nothing can be as satisfactory to a surgeon as to be able to remember that during a difficult operation he has performed to the best of his ability, that he has treated the tissues gently and his associates considerately. Nothing more grievous to the conscientious surgeon that to remember or recall vividly that he has allowed his hands, in carelessness, despair desperation or fatigue, to handle the tissues ungently. He may have been able to repair the damage or not, but its memory will plague him long. It may serve him as it has served me to see and teach how things should not be done, but this is only a sordid substitute for things well done and well taught.

Nothing shows better the victory of the Hiltonian surgery as taught and propagated by apprenticeship, that the disappearance of the old operating "theatre" suited for the dramatic in surgery and its replacement by what is almost an operating temple where the faithful can study essential detail.

Another fact which shows the prevalence of this Hiltonian philosophy is the care now taken by all good surgeons in the skin and subcutaneous tissue. Many a gentle surgeon was often content years ago to apply his principles to the deeper layers only, forgetting the physiology of the skin and the subcutaneous tissues. For a beautiful scar is proof not only of healing without sepsis, but also of healing without any recognizable reaction to repair. It is a guarantee of lasting comfort to the patient and of untainted soil for the man who may have to come afterwards. It is the signature of the gentle surgeon.

This performance of course is made possible by better anesthesia and more highly skilled assistance which have eliminated the need for hurried work so that only its quality need be considered. Their perpetuity is assured by a new school of young surgeons, trained in the use of their hands so they can acquire the touch of an artist, and do the work of a scientist.

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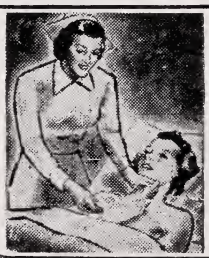

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